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RECESSION OF THE LEVATOR MUSCLE FOR LAGOPHTHALMOS IN EXOPHTHALMIC GOITER

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Several methods have been used in the past to correct the defective closure of the lids incident to the exophthalmos in exophthalmic goiter. Such methods are applicable as a measure of protection to the cornea not only preceding thyroidectomy but after operation as well when the exophthalmos persists.

In general, three methods of approach have been used. The first attacks the lids directly; the second attempts to modify the sympathetic nervous supply of the orbital contents, and the third aims to decompress the orbit. The external tarsorrhaphy advocated by E. Fuchs and the modifications of this operation employed by others correct the deformity to a satisfactory degree. Axenfeld [1] used a looped silk suture. The upper end of a white silk suture was passed along through the upper lid parallel to the margin, and in the same manner the lower end was passed through the lower lid. At the outer canthus the ends crossed and were buried after being tied with sufficient tension to obtain the necessary correction. Jaboulay,[2] in 1896, was the first to perform cervical sympathectomy. Fairly good results were obtained with this procedure. Charles Mayo [3] removed both superior and middle cervical ganglions, as suggested by Jaboulay. In addition, he ligated the superior thyroid vessels through the same incision. As a result of this procedure, the exophthalmos was reduced, and there was a slight drooping of the upper eyelid. Tinker,[4] in 1912, performed a modified Krönlein-Kocher osteoplastic resection of the outer wall of the orbit. He removed fat and resected a portion of the outer wall of the orbit. His

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1. Axenfeld, quoted by Spaeth, E. B.: Am. J. Ophth. **15**:589 (July) 1932.
2. Jaboulay: Bull. Acad. de méd., Paris **38**:139, 1897.
3. Mayo, Charles: The Surgical Treatment of Exophthalmos, J. A. M. A. **63**:1147 (Oct. 3) 1914.
4. Tinker, Martin: The Surgical Treatment of Exophthalmos, J. A. M. A. **59**:989 (Sept. 21) 1912.

end-results were satisfactory. Naffziger and Jones [5] appear to have made a real contribution when they decompressed the roof of the orbit and optic foramen, in this manner releasing the orbital contents and pressure on the optic nerve. They do not advise the operation merely for persistent or stationary exophthalmos. The indications they propose are limitations of ocular movements, loss of vision and changes in the optic nerve. Their results have been good, and there have been no recurrences.

In the following report recession of the levator muscle was performed in each of five patients with exophthalmic goiter. In four of these the exophthalmos persisted following thyroidectomy. The operation was performed with a view to repairing the defective closure of the lids.

REPORT OF CASES

CASE 1.—S. K., a girl, aged 20, was admitted to Mount Sinai Hospital with a history of nervousness, loss of weight and exophthalmos. One year before a two-stage thyroidectomy was performed. The basal metabolism was plus 48. The exophthalmos in the right eye measured 22 mm.; in the left, it measured 20 mm. Vision in both eyes was 15/15. The patient complained of inability to close her right eye. The right levator muscle was receded 5 mm. through the conconjunctival side.

CASE 2.—I. M., a woman, aged 41, with exophthalmic goiter had had a subtotal thyroidectomy in 1924. However, exophthalmos had persisted for the following eight years. She was readmitted to the hospital with an ulcer of the right cornea. It was a central, punched-out ulcer with undermined edges. The exophthalmometer reading (Hertel) was 25.5 mm. in the right eye and 26 mm. in the left. There was, in addition, external strabismus in the left eye. The basal metabolic rate was plus 23. The ulcer was treated for a few days, but without marked improvement. The right levator muscle was receded nasally and mesially 5 mm., and temporally 7 mm. The procedure was carried out from the conjunctival side. The eye was closed for one week. The sutures were then removed. The result was so satisfactory that the patient requested that the left eye be operated on as well. This was done one week later.

CASE 3.—P. B., a man, aged 55, was admitted to the hospital with the complaint of nervousness and a painful right eye. The exophthalmometer reading was 28 mm. in the right eye and 24 mm. in the left. The conjunctiva of the right eye was markedly chemotic and injected. There was a 3 mm. ulcer on the lower part of the cornea. The metabolism ranged between plus 19 and plus 35. Vision in the right eye was 15/70, and in the left eye, 15/50. The right levator muscle was receded 5 mm. through an incision in the skin. The eye remained closed one week. The ulcer was found to be healed. The conjunctiva was less chemotic and injected. The lid covered the entire cornea. One week later the left levator muscle was receded 5 mm.

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5. Naffziger, H. C., and Jones, O. W.: The Surgical Treatment of Progressive Exophthalmos Following Thyroidectomy, J. A. M. A. **99**:638 (Aug. 20) 1932.

CASE 4.—B. O., a woman, aged 47, had a thyroidectomy performed in 1932. Both eyes showed marked protrusion with good motility and the usual lid signs of exophthalmic goiter. Vision in the right eye was 20/20. The media were clear and the fundus normal. The left eyeball was injected, the iris atrophic, the lens cataractous and the vision nil. On April 22, 1933, recession of the levator muscle of the left eye was done and on April 29, recession of the levator muscle of the right eye.

CASE 5.—A. B., a man, aged 47, had a thyroidectomy performed in July, 1930, and a bilateral sympathectomy in February, 1933. He was admitted to the hospital with tremendous exophthalmos of the left eye and beginning endophthalmitis following corneal ulceration with marked chemosis of the conjunctiva and edema of the lids. It was necessary to enucleate this eye. The vision in the right eye was 6/200. The nerve head showed papilledema. Exophthalmos was not measurable on account of the physical condition of the eye. In this case recession was performed as a preliminary step to further treatment. Recession of the levator muscle was done, with marked improvement.

Fig. 1.—The picture on the left shows bilateral exophthalmos; appearance before operation; the center illustration, appearance after recession of the left levator muscle, and on the right, recession of both levator muscles.

A preliminary measurement must be taken to determine the amount of recession necessary. The distance between the free margin of the upper lid and the upper limbus is measured in the vertical meridian of the cornea. Similarly, the measurement is taken of the distance between the free margin of the lower lid and the lower limbus of the cornea. The sum of these distances represents the amount to which the levator muscle should be receded.

TECHNIC

Under local anesthesia (procaine hydrochloride, 2 per cent, without epinephrine) the upper lid is everted and an Erhardt clamp applied with solid blade above. An incision, extending the entire length of the lid, is made through the conjunctiva only at the upper border of the cartilage (fig. 2 *A*). The upper flap of conjunctiva is freed upward from the underlying levator muscle for a distance of 10 mm. (fig. 2 *B*). An incision is now made 1 mm. below the upper edge of the cartilage, through the conjunctiva and cartilage to the fascia separating the levator from the orbicularis muscle (fig. 2 *C*). The cut edge of cartilage to which the levator muscle is attached is grasped and dissected from the fascia upward, for a distance of 10 mm. (fig. 2 *D*). The levator being isolated, three double-armed no. 5 silk sutures on a fairly curved needle three-fourths inch long, are passed through the levator (fig. 2 *E*). The suture is passed from within out, behind the

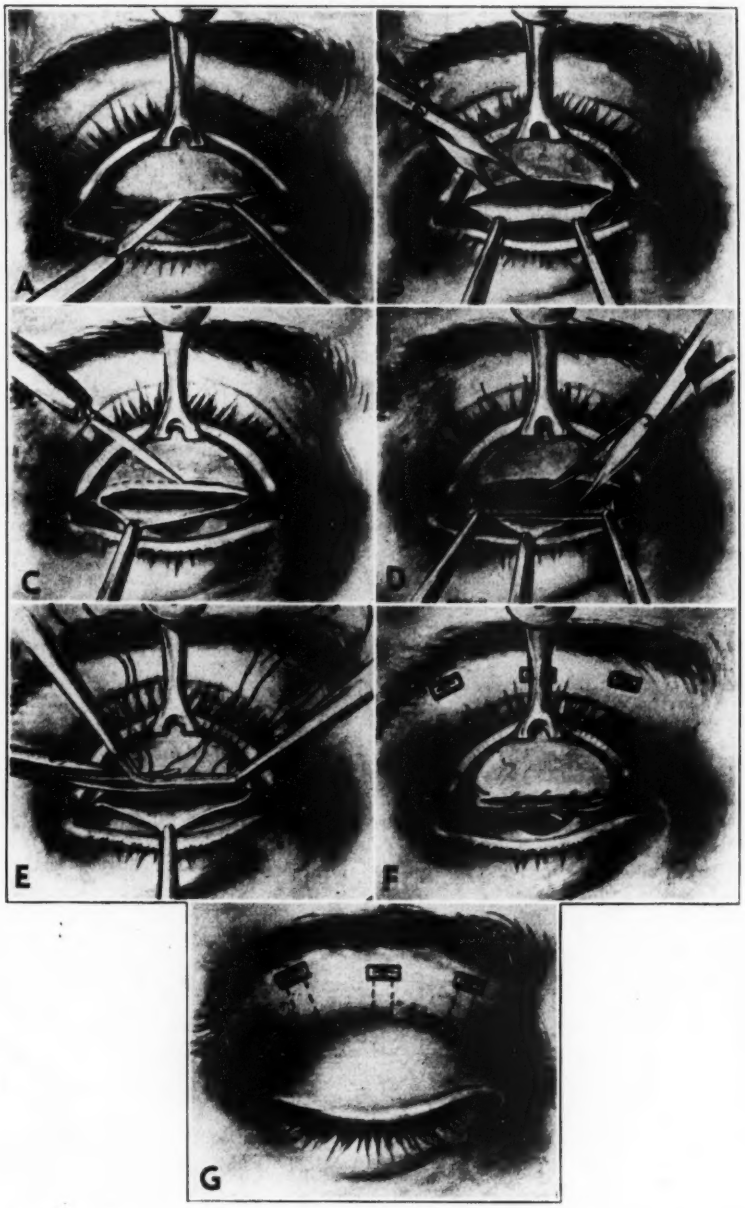

Fig. 2.—Steps in the operation: *A*, an incision is made through the conjunctiva only at the upper margin of the cartilage the entire length of the lid; *B*, the upper flap of the conjunctiva is freed from the levator muscle for a distance of 10 mm.; *C*, an incision is made through the conjunctiva and cartilage 1 mm. below the upper edge of the cartilage the entire length of the lid; *D*, the levator is freed from the underlying fascia upward for a distance of 10 mm.; *E*, three double-armed sutures are passed from below upward, nasally, mesially and temporally, cutting off the millimeter of cartilage before one passes sutures; *F*, the sutures are brought out on the surface of the skin just beneath the brow nasally, mesially and temporally; G, the cut conjunctiva is closed by a running stitch.

strip of cartilage (which is later cut off), and brought out on the skin side immediately below the brow. Three such sutures are passed and tied over pegs (fig. 2*F*). The cut conjunctiva is closed by a running stitch (fig. 2*G*). The eye is dressed in two days, and if the cornea is intact the dressing is left off. The eye is then covered with a Gifford patch made of cellophane. The skin sutures are removed in four days, and the conjunctival sutures in one week. If the cornea has been the seat of an ulcer, the dressing should be postponed for a week.

The operation may be similarly performed from the surface of the skin. By this method, the incision is made through the skin the entire length of the lid, at the upper edge of the cartilage, the globe being protected by a Jaeger horn plate. By blunt dissection, the orbicularis muscle is separated the entire length of the lids, exposing the levator muscle. A lacrimal probe is passed beneath the levator muscle, care being taken not to puncture the conjunctiva. The levator muscle is cut from its attachment and freed upward for a distance of 10 mm. Three double-armed no. 5 silk twisted sutures are passed nasally, mesially and temporally, and are tied on the skin side just beneath the brow. The skin is closed by interrupted sutures. The eye is dressed in two days, and the sutures removed in five days.

COMMENT

In all, eight operations were performed on five patients. The operation for unilateral exophthalmos is more difficult to do, because one has to match the fellow eye. In such a case it may be necessary to make minor adjustments. If ulceration is present, the procedure is modified by leaving the eye closed for one week. Overcorrection did not occur in any case, and all the patients were able to close their eyes during sleep. The exophthalmos was not influenced, but a more normal facial expression was produced. Operation is not indicated if there is serious loss of vision.

ANOMALIES OF THE OCULAR MUSCLES

SYMPTOMS AND DIAGNOSIS, EVIDENCES OF PARALYSIS AND SPASM

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Essential Identity of Symptoms in Underaction and Overaction, However Caused.—Any of the ocular muscles may act insufficiently or to excess, and the overaction or underaction may be due to anomalies of structure, insertion or innervation. The terms paralysis and spasm are sometimes restricted to the innervational anomalies. But, as clinical evidence shows, the symptoms of either overaction or underaction are for the most part the same, no matter what the cause. It is hence convenient to group all of these anomalies in one category, without attempting to distinguish them into structural, insertional and innervational, and to use the terms paralysis and spasm to denote them, no matter what their origin.

This treatment of the subject is especially justified in cases of underaction. An inefficiency of the external rectus due to sheer muscular weakness or to a complete tenotomy resembles in practically all particulars one due to a paralysis of the abducens nerve. This resemblance does not hold to the same extent for the different forms of excessive action. It is true that the contracture that apparently takes place in the antagonist to a paralyzed muscle does simulate closely the conditions presented by a genuine spasm. But a thoroughgoing advancement of a muscle, say the superior rectus, does not produce quite the same effect as a spasm of the muscle. The excessive movement, in fact, is not confined so closely to the field of action of the overacting muscle, but in the case of the superior rectus muscle is distributed more or less over the whole upper field; and, on the other hand, there is produced a restriction of movement in the opponent muscles, much more evident than in the case of pure spasm, and distributed over a large part of the lower field. Yet, notwithstanding this exception, but keeping it in mind, one will find no difficulty and rather a practical advantage in considering all varieties of overaction together.

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† Dr. Alexander Duane died on June 10, 1926. Mrs. Duane has courteously permitted the publication of certain chapters from the book on "Ocular Muscles" on which Dr. Duane was so industriously working and which he had nearly completed at the time of his unfortunate end. These chapters represent the views of an authority who devoted his life to scientific research in a field to which he had so ably contributed.—THE EDITORS.

Paralysis and Spasm.—Each muscle of the eye has one main action, and in most cases also several subsidiary actions as well. Furthermore, there is one special direction of the gaze, different for each muscle, in which its main action increases (fig. 1 of a previous article[1]). This basic fact leads to the following deduction, which is abundantly proved by an enormous mass of clinical evidence and which forms the key to the diagnosis in paralysis and spasm.

In paralysis the movement of one eye is restricted; in spasm the movement is exaggerated in the one special direction of the gaze which represents the field of action of the affected muscle. Moreover, the restriction in the one case and the exaggeration of movement in the other increase in proportion as the eyes are carried in the given direction.

This limitation or excess of movement results in, first, an objectively demonstrable deviation of the eye, and, second, subjective symptoms owing to the limited movement and the deviation. These subjective symptoms are false localization of objects (false projection), apparent movement of objects, diplopia and vertigo. With these may be associated head-tilting or evidences of confused vision, e. g., a tendency to shut one eye. Like the limitation or excess of movement, the deviation and the subjective symptoms increase as the eyes are carried into the field of action of the affected muscle.

These symptoms I shall take up in detail. But before doing so I should like to call attention to one fact of primary importance.

Equivalence of the Effect Produced by Paralysis of One Muscle and by Spasm of Its Associate.—The fact to which I have just referred may be stated as follows:

So far as concerns the relative movements of the eyes and the deviation and symptoms produced, the effect of paralysis of any single muscle of one eye is the same as the effect of the spasm of its associate in the other eye.

To see what this implies, consider a case of paralysis of the right superior rectus muscle. The field of main action of this muscle is found in the diagnostic direction, up and to the right. Hence, when an attempt is made to move both eyes up and right, the right eye lags below the left, and increasing deviation and diplopia develop. But precisely the same evidences and symptoms would be produced if instead of the right eye moving up too little, the left eye should move up too much in the direction of the gaze. Such an excessive movement would be effected by an overaction or spasm of the muscle of the left eye the field of action of which lies up and to the right. That muscle is the left inferior oblique, the associate of the right superior rectus muscle (fig. 1 of a

1. Duane, Alexander: Binocular Movements, Arch. Ophth. **9**:579 (April) 1933.

previous article [1]). A similar relation would hold true for each muscle of the right eye and for its associate in the left.

There is, of course, a difference between the cases in that in a marked paralysis the movement of the affected eye is obviously less and a marked spasm is obviously greater than normal. But so far as the relative movements of the two eyes and the symptoms are concerned, the parallelism is complete. Moreover, in a great many cases of moderate paresis it is actually difficult to tell from the appearances whether one is dealing with an insufficiency in one eye or a spasm in the other.

Finally, it must be added that the correspondence here enunciated furnishes the basis for one method of operative relief in the case of paralytic deviations.

The statement, if put in another form, however, has a wider, indeed, a universal, application. Thus, amended, it may be put as follows:

Paralysis of a muscle in one eye is invariably accompanied by a spasm of its associate in the fellow eye. This spasm becomes manifest as soon as fixation is shifted from the sound to the affected eye.

This is necessarily so. For when the urge is given for any conjugate movement—e. g., a movement to the right—the nervous impulse for that movement is always imparted equally to the right eye and to the left. If the right eye is hampered in its outward movement through weakness of its externus muscle, it will fall behind the left, which is following the object of fixation in a normal manner. Now suppose that by any maneuver fixation is transferred from the left eye to the right. The right eye is now compelled to move out fully, which it does at the expense of considerably added nervous effort. This added nervous effort is necessarily transmitted in equal amount to the left eye too, which being unhampered in its action, will fully respond to it. Accordingly, while the right eye under a great stimulus is with difficulty and effort moving to the right to fix the object looked at, the left eye, being equally stimulated, shoots quite far in the same direction; that is, it acts as though moving under the influence of a spasmodically contracting internus. Thus the paresis of the right externus muscle is coupled with a spasm of its associate, the left internus, as soon as the right eye takes up fixation.

As clinical experience shows, such a secondary spasm of the associate muscle is regularly seen in conjunction with paralyses whenever the paretic eye fixes.

Objective Evidences of Paralysis or Spasm; Lagging or Overshoot; Noncomitant Deviation.—The restriction or excess of action produced by a muscular anomaly may be demonstrated by the tests of the monocular excursions. These will demonstrate a lagging or overshoot, or

more properly will demonstrate a lagging of one eye and an overshoot of the other as soon as the field of action of the affected muscle is entered.

Since one eye lags behind the other and the lagging increases more and more as the eyes are carried into the field of action of the affected muscle, the visual axes tend more and more to differ in direction, and there is thus also an increasing, i. e., a noncomitant, deviation. This deviation is observable by inspection and still better by the screen.

Its character will be determined by the way in which the affected muscle when acting normally moves the eye. Thus if an abductor is paralyzed, the affected eye will not move out as well as its fellow, so that the two eyes will converge unduly; i. e., there will be an esotropia. So if the paralysis affects an adductor, the eyes will diverge or there will be an exotropia; if it affects an elevator of the right eye, that eye will be lower than the left, or there will be a left hypertropia.

The objective signs of muscular paralysis or spasm are thus twofold, viz., noncomitance (i. e., lagging or overshoot) and deviation. The

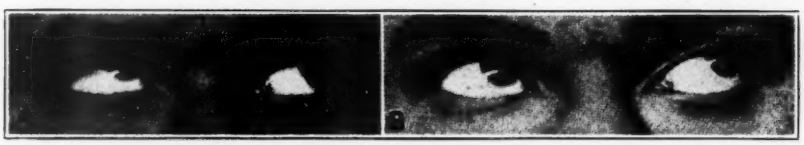

Fig. 1.—*A*, paralysis of the left superior rectus muscle. When the eyes are carried to the left and the left eye is fixed, the right shoots obliquely up. *B*, same case after tenotomy of the right inferior oblique muscle. There is no up-shoot in looking to the left.

noncomitance and deviation differ in amount according as the sound or the affected eye fixes, being denoted as primary in the former case, and as secondary in the latter. In a case of paralysis the primary noncomitance (lagging of the affected eye) is less than the secondary noncomitance (overshoot of the sound eye); and the primary deviation, shown by the screen test when the sound eye fixes, is less than the secondary, when the paretic eye fixes. In spasm just the reverse is true; the overshoot of the affected eye when the sound eye fixes being greater than the lagging of the good eye when fixation is shifted to the other; and the primary deviation, when the sound eye fixes, is greater than the secondary. In other words, the more powerfully acting eye makes a more extensive excursion and deviates more behind the screen than its fellow, and this happens no matter whether it or the other is the one affected. This is but another illustration of the equivalence which exists between paralysis in one eye and spasm in the other.

The secondary deviation of the sound eye shows itself not only when this is screened but also when for any other reason the paretic eyes takes up fixation.

This happens, for example, when the patient looks so far to one side that the view of the object is cut off from the sound eye by the nose. The sound eye in that case shows a sudden marked deviation (fig. 1 *A*). Again it often happens, especially in congenital paralysis, that the paretic eye is used habitually for fixation, so that the sound eye deviates in a spasmodic way, even when both eyes can see the object. Or, fixation may alternate between the two eyes, in which case the difference between the small primary and large secondary deviation becomes at once apparent.

Subjective Symptoms of Paralysis and Spasm.—The subjective symptoms of paralysis and spasm are the following:

Sense of discomfort in moving the eye.
False projection.
Apparent movement of objects.
Varying diplopia.
Vertigo.
Confused vision.

Diplopia is present only when both eyes are used together. The other symptoms are present whether the patient is looking with the affected eye alone or with both eyes, but the vertigo and confused vision are specially marked in binocular vision.

To these symptoms must be added those produced by the condition causing the paralysis. Such are the headache in paralysis due to syphilis or meningitis, the increased intracranial pressure in paralysis due to cerebral and cerebellar tumors, the evidences of disease of the other nerves, especially the optic nerve and its connections (optic neuritis, restriction of the field) and the facial nerve (facial paralysis), and hemiplegia, especially in diseases of the cerebral peduncles and the pons.

Sense of Strain Produced by Moving a Paretic Muscle.—The very act of exerting a paretic muscle excessively in order to make the eyes move may give rise to discomfort or even pain. It is rather surprising, indeed, that the symptom is not more often remarked. Probably the reason is that the patient does not often voluntarily attempt movements of any extent that call the affected muscle into play.

Muscular strain may perhaps be accountable for the asthenopia in near work produced by postoperative insufficiencies of the internal recti muscles, and by moderate pareses of the superior oblique or the inferior rectus muscle.

False Projection.—Notions of the position of external objects are based partly on the visual impressions that these objects set up and partly on the sense of effort required in order to turn the head or eyes toward the object (postural impressions). Usually these postural impressions dominate. If either set of impressions is interfered with, judgment of the apparent position of objects is rendered faulty.

An interference with the postural impressions is produced whenever there is a paralysis of one of the ocular muscles. Suppose, for example, that a man with paralysis of the right external rectus muscle is called on to sight with the right eye an object situated 10° to his right. Ordinarily this would take a moderate amount of muscular exertion, corresponding to which there would be expended a certain nervous effort. This nervous effort would from experience be associated in his mind with a rotation of the eye 10° to the right. But now as the muscle is weakened he has to make a much greater effort—say twice as great—in order to make the 10° rotation. This greater effort would mean for him ordinarily a greater turning of the eye, and, in consequence, he locates the object not 10° but 20° to the right. On the other hand, when he looks to the left, in which direction the muscles act normally, he needs only to make the usual effort and hence projects as he normally does. Objects on the left side, then, are seen in their true place, while objects on the right side are all projected too far to the right.

A similar reasoning holds in the case of paralysis of the other muscles. In paralysis of the right internal rectus muscle, an object to the left would look too far to the left; in paralysis of an elevator muscle, an object situated above would look too high, and in paralysis of a depressor muscle, an object situated below would look too far down. In other words, in any case of paralysis, as soon as the affected eye is carried into the field of action of the paralyzed muscle, the object of fixation appears to be beyond its true position, i. e., is located too far out, in the direction in which the eye is moving. Accordingly, if the patient attempts to point to it he overshoots or points past it.

In overaction (spasm) of an ocular muscle the reverse holds good. In this case little effort is required in order to make the eye move considerably, and this small effort is translated to mean little motion. As soon, then, as the object is carried into the field of action of the paralyzed muscle, it seems to fall short of its true position; i. e., it is located nearer the midline than it really is. Moreover, if the patient attempts to point to it, he undershoots the mark.

It is noted that in paralysis even an object which is straight ahead will appear deflected somewhat to the side to which the paretic muscle normally moves the eye. For in paralysis of the right external rectus, the muscle, unless it makes an extra exertion, balances but imperfectly the normal pull to the left made by the internal rectus muscle, and hence is deflected somewhat to the left. It is only by the interposition of a definite stimulus to move to the right that it can be kept straight on the object, *A*, straight ahead. The extra stimulus is interpreted by the patient as signifying that *A* is not straight ahead but is located somewhat to his right. If he makes no effort to move the eye farther to the right, the position of objects in the right field will be judged by

the visual relations to *A*, i. e., they will all be deflected equally and slightly to the right. This of itself would occasion little trouble, if it were not for the fact that as soon as the gaze is shifted a sudden change in the projection takes place which causes two other disagreeable symptoms, namely, an apparent movement of objects and a rapidly varying double vision.

A patient seeking to locate solely with the aid of the paralyzed eye an object in the field of action of the paralyzed muscle will make wrong judgments as to its position. The object, in fact, seems displaced in the direction in which the paralyzed muscle if normal would move the eye. This leads the patient with the pointing test to shoot past the object which he is trying to touch. Again, if he tries to walk toward a point, using his paralyzed eye alone, he will go straight if the object is not in the field of action of the paralyzed muscle; otherwise, he takes a wavering and zigzag course, first bending his steps too far to the right, then recognizing his mistake and correcting it; then deviating anew to the right, and so on.

The explanation of this occurrence is similar to that which has been given for binocular diplopia. The object is falsely localized because the patient is in error in regard to the position which his eye occupies. When the patient with his paralyzed right eye sights the object *o*, which is placed somewhat to the right of him, i. e., looks at it in such a way that it forms an image upon the fovea centralis, *f*, he can accomplish this only by the strongest possible innervation of his paralyzed externus. Now, the ideas which one has with respect to the position of one's eyes depend on one's sensations with regard to the innervation of the individual muscles. The patient, therefore, is compelled to believe that the right eye is standing in the most extreme position of right lateral rotation, like the eye represented by the dotted line in figure 2, because he has sent an impulse for a rotation to this amount into the right externus muscle, and he cannot know that the latter, owing to the impairment of the conduction, only partially obeys this impulse. He hence proceeds on the assumption that the right eye is turned very strongly to the right, and that consequently its fovea is at f_1; he is, therefore, also compelled to believe that the object, the image of which is formed at the fovea lies opposite f_1—i. e., at o_1, and he hence sees the object too far to the right.

Confused Vision and Vertigo.—The apparent movement of objects and the varying diplopia produced by the false projection often, especially in recent cases of paralysis, give rise to vertigo. Still more often and more persistently they cause confusion of sight. This is especially great when the shift in place of the images is considerable, so that now they are close together, now farther apart. This occurs particularly in recent paralyses before compensating changes have set in. The

diplopia is also confusing when the images are quite close together or even overlap. Anything, therefore, that will separate them farther and also render the diplopia more comitant will be a relief.

It is to be remarked that the changes which so frequently take place in the pathologic conditions act precisely to effect these beneficial results; i.e., they separate the double images and render the deviation more constant.

Evidence of Attempt to Relieve the Subjective Symptoms, Monoblepsia.—Another means that the patient has of obviating confusion, vertigo and diplopia is shutting one eye. This will at once relieve the diplopia, no matter which eye is shut, but does not necessarily relieve the other symptoms, even the confusion and vertigo, unless the patient

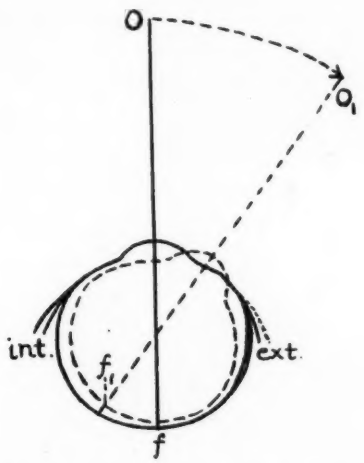

Fig. 2.—False orientation in paralysis of the right externus muscle.

happens to close the affected eye, which, in fact, he usually does. The habit of closing one eye, which may be noted even in infants affected with paralysis, is called monoblepsia.

Attitude of the Head in Paralysis; Ocular Torticollis.—Still another means of obviating diplopia is by turning the head. This symptom deserves special attention, not only because of the diagnostic significance attributed to it, but also because not infrequently it is so disfiguring or produces so much discomfort that it calls for relief on its own account.

On this subject a good deal has been said in special treatises that seems to have been dictated by theory rather than by observation. The so-called characteristic attitudes of the head in paralysis are not very often seen. In fact, in cases of the same kind one finds the patient turning his head sometimes in one way, sometimes in another. Following are my observations on this point.

Turning the head in paralysis takes place in three different ways:

1. The head may be turned in the direction in which the muscles, when acting normally, move the eye. Thus in paralysis of the right external rectus muscle, the head is turned to the right. The right eye in that case, when sighting an object straight ahead, is really turned to the left, i. e., away from the field of action of the affected muscle or into a quarter of the field where even with the paralysis there is no deviation. This is the method commonly followed in paralysis of a lateral rotator, the head being turned to the right in paralysis of the right external or the left internal rectus muscle and to the left in paralysis of the right internal and the left external rectus muscle. So also in paralysis of an elevator, especially when very marked, one often sees the head turned up (tipped back), and probably less often in paralysis of a depressor one sees it turned down (tipped forward).

2. In the case of paralysis of an elevator or a depressor, the head may be turned toward the side, right or left, on which the main action of the muscle (elevation or depression) is normally at its maximum. In other words, in paralysis of a right-hand elevator or depressor (right superior and inferior recti muscles and left superior and inferior oblique muscles) the head would be turned to the right; in paralysis of the other vertically acting muscles (left-hand elevators and depressors) it would be turned to the left. This is not frequently done.

3. Also in the case of paralysis of an elevator or a depressor the head may be tilted to one shoulder. This is a frequent attitude, particularly in the more moderate degrees of paralysis. Commonly, but not universally, one finds in a paralysis of the superior rectus muscle the head tilted to the side of the paralyzed muscle, and in paralysis of the superior oblique muscle it is tilted toward the opposite side.

In not a few cases two or even all three of these different types of turning the head are combined.

Tilting the head to the side in paralysis is sometimes so marked as to constitute a false torticollis (ocular torticollis), which is distinguished from true torticollis primarily by the fact that the head straightens, often immediately, if the paretic eye is bandaged, and also by the fact that when the head is straightened and both eyes are left open there is a marked vertical squint which disappears or grows very much less when the head reverts to the tilted position. In spite of these diagnostic signs, the patient is often for months treated by orthopedic appliances, and naturally not only with no benefit, but even with additional discomfort.

This is illustrated in the following cases:

CASE 1.—*Paralysis of the Right Superior Rectus Muscle (Congenital); Marked Tilt of the Head, Relieved by Tenotomy of the Left Inferior Oblique Muscle.*—

Hortense S., aged 3 years and 9 months, had had a tilt of the head to the right shoulder since the age of 4 months. Since the age of 6 months she had been treated for torticollis with bandages and electric massage. The mother observed that when the head was straightened the left eye deviated up, and acting on this hint bandaged the child's left eye, whereupon the head became straight.

The head tilted sharply to the right shoulder (fig. 4), and generally rotated somewhat to the right. With the head held straight, LH (Sc) > 10°, and >> in Er and especially in Eu & r (marked upshoot of the left eye in these directions). Ed & r and Ed & l movements were fairly comitant. When the head was tilted, LH << to 5°.

Tenotomy of the left inferior oblique muscle was performed. Three weeks later, the head was nearly straight (fig. 5). LH was reduced to a few degrees. Six years later LH was only minimum (Sc). The head was straight.

CASE 2.—*Paralysis of the Right Inferior Rectus Muscle (Congenital); Tilt of the Head; Relief by Operation.*—Lilie N., aged 22, had tilt of the head since the age of 4 or 5. There was no spontaneous diplopia. Asthenopia and headache were

Figure 3

Figure 4

Figure 5

Fig. 3 (case 1).—Position of the head at 7 months.

Fig. 4 (case 1).—Position of the head at 3 years.

Fig. 5 (case 1).—Position of the head one week after tenotomy of the inferior oblique muscle.

produced by stenographic work. She had worn an apparatus to correct the deformity.

The head was tilted far over to the left shoulder. Vision was 15/15 in both eyes. There was slight hyperopia, but no astigmatism. With the head in the usual tilted position there was little or no vertical deviation; with the head straight, RH (Sc) and DR 17-20° with some DX. Deviation was somewhat greater when the right eye fixed. Excursion tests showed the following:

	Right Fixing	Left Fixing
Eu & r	L. lagged moderately	R. deviated up but no marked upshoot
Eu & l	L. lagged moderately	R. deviated up but no marked upshoot
Ed & r	L. far down (cornea almost out of sight); R. down with difficulty	R. did not go below horizontal
Ed & l	L. much below, but not so greatly as in Ed & r	R. down moderately

Diplopia was seen on a tangent curtain (fig. 6).

Treatment consisted in advancement of the right inferior rectus muscle. One week later this was still very marked RH. Tenotomy of the right superior rectus muscle was performed. Six months later there was single vision over a large portion of the field, with DL in the upper field (evidently due to postoperative weakness of the right superior rectus muscle) and DR with DH in the extreme lower field (40° below the fixation point). The patient held the head straight. The occipital headaches, which formerly were marked, had disappeared. When examined five years later, the position was good and the motor conditions were quite satisfactory in spite of marked residual insufficiency of the right inferior rectus muscle (for the field of diplopia see fig. 7).

In this case there was evidently a primitive, probably congenital, paralysis of the right inferior rectus muscle, with consecutive changes carrying the diplopia

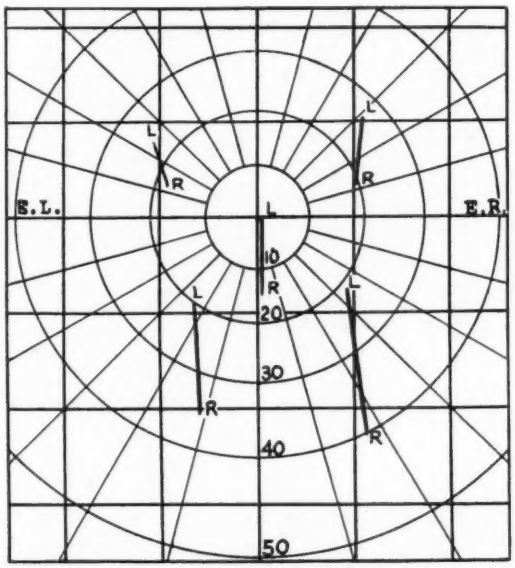

Fig. 6 (case 2).—Diplopia in paralysis of the right inferior rectus muscle with secondary changes, before advancement of the muscle. There is marked DR, with a tendency to increase in Ed & r, but extending into the upper field. There is no single vision anywhere. Note the relation clearly shown by excursion tests.

well into the upper field. The headaches, it is not unlikely, were due to the constrained attitude of the head.

As Bielschowsky [2] pointed out, ocular torticollis was recognized in 1871 by Cuignet, and more fully studied by Landolt in 1890. That the tilt of the head is assumed in order to obviate the deviation is clear from the fact that it disappears either when the deviating eye is covered or when the deviation is relieved by operation. How it obviates the deviation has been a matter of some controversy.

2. Bielschowsky, A., in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1910, vol. 8, chap. 22, pt. 1.

Bielschowsky [2] expressed the belief that the reduction of the deviation is brought about by the effect of the attitude on the torsion action of the muscles involved. According to his theory, in paralysis of the superior oblique and of the superior rectus muscle, a reduction should be produced by a tilting of the head to the side opposite the affected eye; while in paralysis of the inferior rectus and inferior oblique muscles the tilting, to be of service, should be toward the side of the affected eye. And, indeed, he found in a great many cases of paralysis of the superior oblique muscle that tilting of the head to the opposite side invariably reduced the deviation. I too have found that in most cases of paralysis of the superior oblique muscle the tilt of the head that the patient assumes to correct the deviation is to the opposite side. But this is by no means invariably the case. Moreover, as I have quite regularly found, paralysis of the superior rectus muscle

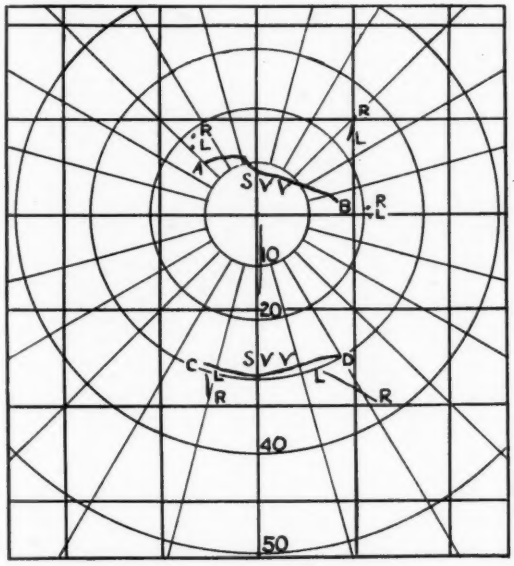

Fig. 7 (case 2).—Diplopia in paralysis of the inferior rectus muscle after operations. Same case as shown in figure 6 five years later. Diplopia after advancement of the right inferior rectus muscle and tenotomy of the right superior rectus muscle. Note the large area of single vision (banded in cases AB, CD). In the upper field, especially in Eu & r, DR is indication of an insufficiency (post-operative) of the right superior rectus muscle. In the lower field, DR is indicative quite clearly of a residual paresis of the right inferior rectus muscle. The conditions were practically the same five years later.

is in the great majority of cases associated with a tipping to the same side, instead of a tilting to the opposite side as Bielschowsky's theory demands.[3] Again, in paralysis of the inferior rectus muscle the tilting is sometimes to one side and sometimes to the other, instead of being always to the side of the paretic muscle as it should be if this hypothesis was true.

3. For this reason I think that Bielschowsky's criticism of Nieden's case (Bielschowsky,[2] p. 146) is not justified.

If postural projection governs in cases of paralysis as it does in normal cases when the head is tilted, the following results would ensue:

In conditions associated with DR (paralysis of the right superior oblique or the inferior rectus muscle or of the left superior rectus or the inferior oblique muscle) the vertical diplopia should be reduced by tilting the head to the right if the DR is associated with DX (fig. 8), to the left if the DR is associated with DH, and either to the right or the left if there is no lateral diplopia.

In conditions associated with DL (paralysis of the right superior rectus or the inferior oblique muscle or of the left inferior rectus or superior oblique muscle), the vertical diplopia should be reduced by tilting the head to the right if the DL is associated with DX, to the left if the DL is associated with DH and either to the right or the left if there is no lateral diplopia.

Tilting of this sort may be called tilting according to rule.

If for any reason the patient should ignore the information afforded in postural projection which would assure him that his head was tilted and should project according to visual impressions alone, the very reverse of the foregoing statements would apply. In this case, in order to obviate the diplopia, he would tilt the head contrary to rule.

In cases that I examined, some patients tilted the head according to rule, some tilted it the contrary way, and in some the finding was uncertain because there

Fig. 8.—*L* denotes the left-eye image; *R*, the right-eye image. Hence *A* denotes crossed and right diplopia (DX and DR); *B*, homonymous and left diplopia (DH and DL), *C*, homonymous and right diplopia (DH and DR), and *D*, crossed and left diplopia (DX and DL). If the ordinary rules of projection in tilting the head prevail, a side tilt to the right will diminish the vertical diplopia in cases *A* and *B*, and a tilt to the left will diminish it in cases *C* and *D*.

was little or no lateral deviation. The cases contrary to rule seemed to occur especially in congenital paralysis. Whether the anomalous tilting was due to a peculiar projection (predominance of visual over postural impulses) seems to me uncertain.

Extent of Field Through Which Evidences of Paralysis and Spasm Are Manifested.—The objective and subjective evidences of paralysis and spasm increase progressively as the eyes are carried into the field of action of the affected muscles. But they are also evident when the eyes are directed straight ahead, and in cases of well marked paralysis even when the eyes are carried some little distance into the other fields. Thus the effects of paralysis of the right external rectus muscle are evident not only over the right, but to a varying degree also over the left, half of the field of fixation. Similarly, the evidences of a fairly complete paralysis of the right superior rectus muscle are found not only in the upper right-hand quadrant of the upper field but to a diminishing

degree also in the upper left-hand quadrant and also in the lower right-hand quadrant. Indeed, the extension into the lower field is often quite considerable. This extension is due partly to the fact that a muscle begins to some extent to act soon after its opponent starts to relax, i. e., while the eyes are still well within the field of action of that opponent, and a marked impairment of its activity will thus make itself manifest here too. To a certain extent also the extension, particularly when great, is due to secondary changes taking place in accordance with the law of equalization, the operation of which will be described in some detail presently.

Variation in Symptoms.—The symptoms in paralysis vary greatly in individual cases. In slight paralyses it often happens that little complaint is made of diplopia or vertigo, but, particularly if a depressor is affected, the patient has confusion and asthenopia in reading, book-keeping or other near work. But in more marked cases, particularly in those that are recent, the characteristic symptoms—diplopia, confusion, false projection and vertigo—are usually prominent and often very distressing. The symptoms are especially disturbing when fixation shifts suddenly from the sound to the paretic eye.

If the paralysis gets well comparatively soon, the symptoms that it produces disappear, and normal binocular vision is restored; if, on the other hand, the paralysis persists for a long time, the symptomatic picture changes as follows: 1. The mistakes in projection, particularly as they make their appearance in the pointing test, gradually cease; the patient learns by experience that the impulses of innervation for his paralyzed eye correspond to a much slighter action than those for the sound eye, and by taking account of this fact he once more forms a correct judgment of the situation of objects. 2. The diplopia may disappear through suppression, although such suppression is rarely complete and in many cases does not occur. But, though not actually eliminated, the diplopia after a time gets to be much less obtrusive and annoying. 3. As will be pointed out presently, it often happens that, owing to contracture of the antagonists of the paralyzed muscle and other changes, the deviation and diplopia increase in amount and become apparent not only in the field of action of the paralyzed muscle, but also in the field of action of its antagonist, and may even extend so far over the whole field of fixation that the paralytic strabismus gets to resemble an ordinary comitant squint. In proportion as it does so, the symptoms grow less disturbing; for, as already stated, it is always the case that, other things being equal, a comitant deviation will give less trouble than will one that is noncomitant.

Development and Course.—Many cases of paralysis and spasm develop suddenly, reaching their full height within one or two hours.

Others develop more or less gradually. An attack of paralysis may have premonitory symptoms, especially headache. This is seen particularly in syphilitic paralyses, and in these cases the headache may disappear as soon as the paralysis sets in. (A similar alternation of headache and paralysis is seen in ophthalmoplegic migraine, which is not usually of syphilitic origin.)

The course of a paralysis is regularly chronic. Even in the most favorable cases it lasts for weeks, and often indefinitely. It often shows considerable variations in intensity, and, even after an apparent recovery, may recur either in the same or, more usually, in an altered form. Recurrences are specially frequent in the syphilitic paralyses. Recurrences which are spread over many years and in which each attack is marked by an exacerbation of the paralysis mark the special feature of ophthalmoplegic migraine.

It is obvious that the character of the onset, the course and the duration must depend mainly on the cause and on the extent and gravity of the lesions which that cause produces. The general tendencies shown in the two main classes of paralyses, the congenital and the acquired, are:

(*A*) Course in Congenital Paralyses: Congenital paralyses are usually caused by structural or insertional anomalies which are due either to developmental errors or to traumatism. These anomalies show little or no tendency to change, nor do they seem often to engender secondary changes in the synergists or opponents of the muscles affected. On the other hand, they not infrequently are followed by convergence or divergence anomalies, the development of which undoubtedly has the effect of making the diplopia caused by the paralysis less disturbing. In this way they give rise to a secondary convergent or divergent comitant squint.

(*B*) Course in Acquired Paralyses: Acquired muscular deviations, whether due to anomalies of innervation or of insertion, tend to become comitant. This is in accordance with the law already mentioned, by which deviations in general tend to become more comitant and hence less disturbing to the patients. As a first step in this process a varying or perhaps intermittent spasm develops in certain of the unaffected muscles. The effect of this spasm is to extend over a larger part of the field the diplopia which has been at first confined to a restricted area. The spasm may affect:

(*a*) A synergic muscle in the same eye. Thus in paralysis of the right superior rectus muscle there may be a sort of compensatory spasm of the right inferior oblique muscle. The resulting condition is quite like that produced by simultaneous paralysis of both superior recti muscles, and differs only in that the diplopia in the left upper field fluctuates

considerably and irregularly from one time to another. So, too, in abducens paralysis one sometimes finds an associated spasm of the other two abductors (the two obliques) in the same eye. This causes the eye to move by zigzag jumps a little farther out than the externus alone can carry it. If the eye is above or below the horizontal plane, the amount of additional movement thus obtained may be rather large.

(*b*) The direct antagonist of the affected muscle. The following case is an instance of this by no means infrequent condition.

CASE 3.—*Paralysis of Inferior Rectus Muscle; Varying Spasm of Superior Rectus Muscle of Same Eye.*—H., a man, aged 29, had had vertigo, blurring of sight and diplopia for the past two years. There was no history of syphilis. He presented all the evidences (by inspection, screen and diplopia tests) of a well marked paresis of the right inferior rectus muscle. DR >> in Ed & r, to 6-7° (= 14△); << to 0° in Ed & l. There was moderate DX everywhere, increasing in Ed & l. DR prevailed, although to a less extent in the upper field; most marked in Eu & r. This diplopia in the upper field varied greatly, and was partially superable by voluntary effort.

Here there was evidently a spasm (variable in amount) of the right elevators, and particularly of the right superior rectus muscle (shown by the fact that the vertical diplopia increased upward and to the right). The vertical diplopia in the upper part of the field might, it is true, have been due to a paresis of the elevators of the left eye; but the variable character of the diplopia and the fact that it was superable by voluntary effort negative this supposition.

Later, such an intermittent and varying compensatory spasm tends, as I find, to be replaced by more permanent conditions which tend to make the deviations more and more comitant. Such conditions are a gradually developing contracture of the antagonists when the affected muscle is too weak, and a weakening of the antagonists when the affected muscle is too strong. It is thus, probably, that many cases of comitant esotropia and exotropia are produced; and most of the cases of comitant vertical strabismus that I have met with apparently developed in this way.

Thus paralysis of the right elevators, when not of congenital origin, is regularly followed by spasm of the right depressors, causing the right eye to stand lower than the other in all portions of the field of fixation. This process continuing and the spasm developing into a confirmed contracture, the difference in height between the two eyes becomes equal for all directions of the gaze; the limitation of movement of the right eye upward is compensated for by a corresponding increase in its movements downward, and the picture of a typical comitant right hypotropia is produced.

If but one of the elevators, e. g., the superior rectus muscle, is paretic, the spasm is apt to be confined, in the first instance at least, to the corresponding depressor, i. e., the inferior rectus muscle. In this case the deviation will become half-comitant, that is, comitant for movements up and down but not for lateral movements. Thus, if the right superior rectus were the paretic muscle, there would be first a right hypotropia, very marked in the upper right-hand quadrant

of the field of fixation and diminishing rapidly, both when the eyes were carried down and when they were carried to the left. Later, when spasm of the right inferior rectus muscle had developed, the right hypotropia would become pronounced in the lower right-hand quadrant also, and ultimately so much so that whether the eyes were carried up or down the right eye would always stand about the same distance below the left. But in both the upper and in the lower portion of the field the right hypotropia would still increase as the eyes were carried to the right and diminish to zero as they were carried to the left; so that even under these circumstances the deviation would not be comitant for lateral movements.

Cases in which a comitant (and even an alternating) lateral squint has developed out of a paresis of the externus, followed by contracture of the internus muscle and later by partial restoration of the power of the externus muscle, have been reported by Spicer.[4] I have observed analogous cases, particularly one in which a partial oculomotor paralysis with secondary contracture of the externus had resulted in a nearly comitant exotropia.

Usually, however, even when this sort of extension has been carried quite far one can make out the characteristic increase of the deviation in some special quarter of the field and thus recognize its paralytic origin.

In some cases the contracture of the antagonist may persist after the paralysis itself is cured and may thus prevent the restoration of binocular vision.

DIAGNOSIS OF PARALYSIS AND SPASM

What to Determine in Cases of Paralysis and Spasm; General Plan of Investigation.—In examining a case of paralysis or spasm one should have in mind two things: first, to determine the muscle or muscles affected; second, to ascertain the disturbance that the muscular anomaly causes in ordinary positions of the gaze. The first object is effected by ascertaining the behavior of the eyes and the deviation and diplopia that develop when the eyes are carried in their limit of action in the six diagnostic directions. The second object is accomplished by ascertaining the amount and kind of deviation and diplopia present when the eyes are directed straight ahead or are deflected but a few degrees from the straight forward position. The second determination is very important, since it shows how much and in what way the muscular anomaly is interfering with ordinary useful vision. But, it must be repeated, the actual diagnosis of the muscle affected can be made only by the first tests, in which the eyes are compelled to make excursions of an extent never normally employed.

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4. Spicer: Roy. Ophth. Hosp. Rep., London **14**:1, 1895.

The diagnosis, then, is determined by tests conducted along the following lines:

Tests for Diagnosis of Paralysis and Spasm

MAIN TESTS

Determination of deviation and diplopia in primary position
Binocular excursion tests
Objective: Comitance and screen comitance tests; screen deviation in outlying position
Subjective: Mapping of field of diplopia

SUBSIDIARY TESTS

Monocular tests:
Range of monocular excursions
Projection test
Tests of torsion

The outline given will suffice for the determination of the muscle or muscles affected by the paralysis. In addition, for purposes of treatment one must ascertain those facts that will enable one to determine the cause of the paralysis and the site of the lesion producing it. Such facts include the vision and field of vision, the conditions of the fundus and especially of the optic nerve head; evidences of increased intracranial pressure and intracranial inflammation afforded, for example, by lumbar puncture; evidences of paralysis of other cranial nerves, e. g., the facial and fifth nerve; evidences of body paralysis or ataxia, and disorder of the reflexes. Furthermore, evidences of syphilis, tuberculosis, tumor of the brain, intracranial hemorrhage, toxis, influenza, diabetes, or any other of the numerous possible causal conditions are afforded either by the history or by examination.

Determination of Deviation and Diplopia in Primary Position; Primary and Secondary Deviation.—The deviation and diplopia for distance and near vision in the straight forward direction of the gaze are determined by the screen and diplopia tests (especially the red glass). Special note is made of the patient's ability to overcome the deviation, i. e., whether in the primary position this deviation is merely a heterophoria or whether it is a squint, and, if the latter, which eye habitually fixes. In this connection, one must remember that in paralytic squint the deviating eye is not necessarily the one affected with the paralysis. The variations in the amount of deviation from time to time should also be carefully ascertained as throwing light on the progress of the condition.

It will be found that patients with paralytic conditions of the ocular muscles differ greatly in their ability to overcome the deviation. Instances in which the ability was rather extraordinary have been cited. On the other hand, in some cases paretic deviations of only 1° or 2° cannot be overcome at all.

A deviation found in the primary position has of itself no diagnostic significance. This is shown by the following statement:

In determining the deviation and diplopia, whether in the primary position or in outlying directions of the gaze, it is well to note and, if necessary, measure the amount shown first when the right and then when the left eye fixes (i. e., to determine the primary and secondary deviation). With the screen test, the prism placed before the left eye abolishes all perceptible movement, measures the deviation when the right eye fixes, and vice versa. The corresponding difference in the diplopia when each eye fixes can be determined in the way indicated in table 1.

TABLE 1.—*Deviation and Diplopia in Primary Position*

Kind of Deviation and Diplopia	Paralysis of	Spasm of	Comitant Deviation
S and DH	R. or L. externus rectus, but also of any muscle of either eye	R. or L. internus rectus, but also of any muscle of either eye	Divergence insufficiency or convergence excess
X and DX	R. or L. internus rectus, but also of any muscle of either eye	R. or L. externus rectus, but also of any muscle of either eye	Convergence insufficiency or divergence excess
RH and DR	R. inferior rectus or superior oblique; L. superior rectus and inferior oblique	R. superior rectus or inferior oblique; L. inferior rectus or superior oblique	Comitant hyperphoria
LH and DL	R. superior rectus and inferior oblique; L. inferior rectus and superior oblique	R. inferior rectus and superior oblique; L. superior rectus and inferior oblique	Comitant hyperphoria

Regularly, the eye which shows the greater deviation behind the screen or which requires the greater prism over it to effect correction is overacting as compared with its fellow. Whether this relative overaction is due to a real excess of energy in this eye or to a deficiency of power in the other can be determined by taking the range of excursion in the latter. If this range is less than normal, then this eye must be regarded as paretic, and the excess of action in the other eye is relative only, i.e., represents simply the extra stimulation imparted to a normally moving eye when its paretic associate is urged to action. The primary deviation, i. e., the deviation when the sound eye fixes, is regularly less than the secondary deviation in cases of paralysis and greater than the secondary deviation in cases of spasm. But to this rule there are not a few exceptions, so that without any apparent reason the primary deviation in paralysis is greater than the secondary deviation.

Case 4 illustrates the occasional diagnostic significance of the relation between the primary and secondary deviation.

CASE 4.—*Secondary Deviation Proving an Overaction Produced by Advancement and an Underaction Produced by Recession.*—Gustova M., aged 16, had had alternating esotropia since the age of 4 years. Fourteen months prior to examination operation (evidently advancement or resection) was performed on the right external rectus muscle; three months previously operation (probably a recession) was performed on the right internal rectus muscle.

Vision in the right eye with +150 was 19/20; that in the left eye with —0.37 cyl. 180°, 19/15. There was alternating esotropia both for distant and near vision. Examination showed: S (Sc) 16°; S′ (Sc) 21-25°; about the same prisms and red glass and amblyoscope. There was no vertical deviation. In Er S (Sc) was more when the left eye fixed than when the right eye fixed; in El, the reverse was true. All movements were normal, except that with the left eye in Er > n; in El < n by 1 or 2 mm.

Here when the eyes were turned to the right the deviation was greater when the left eye fixed, indicating that the right eye was acting more vigorously than the left, in spite of the fact that the left eye in this direction turned excessively far in, i. e., was not hampered in movement. The relative overaction of the right eye in looking to the right was thus a real overaction, such as could be attributed only to excess of energy on the part of the external rectus muscle. This excess was imparted to it evidently by the operation (advancement). In a similar way, the fact that in looking to the left the deviation was greater when the right eye fixed indicated that the right eye was, relatively speaking, underacting. This relative underaction could not be due to an absolute excess of action on the part of the left eye, for, as the tests showed, this eye was really hampered in its leftward movement. Hence the underaction of the right eye in looking to the left must have been not simply relative, but absolute, and due no doubt to the weakening of its internal rectus muscle produced by operation.

Lagging and Overshoot of Either Eye in Different Directions of the Gaze.—The application of the comitance and screen comitance tests affords the diagnostic indications given in table 2 in cases of paralysis or spasm.

Diplopia and Deviation in Different Directions of the Gaze.—In a case of paralysis or spasm the deviation behind the screen and the diplopia increase characteristically in some one of the six diagnostic directions. Theoretically, the two should be identical in amount, and, as a matter of fact, the correspondence between the two is generally striking. Where differences exist, it will almost always be found that the diplopia shows the more capricious and anomalous variations. These variations are due either to the patient's errors in judgment or to the fact that he is varying the actual distances between the double images by unconscious efforts of adjustment. The test for diplopia, therefore, should always be confirmed by the determination of screen deviation and by the comitance or screen comitance tests.

The diagnosis of paralysis by the double images and screen deviation can be readily and simply accomplished by an analysis which shows in succession, first, which group of muscles (lateral rotators, elevators or depressors) is affected; next, which pair of associates in the group, and, lastly, which muscle in the pair.

Thus, if there are diplopia and deviation which are mainly lateral and which increase fast in looking to the right or the left, the paralysis must affect a lateral rotator. If the diplopia increases to the right the paralysis affects a right rotator (right externus, left internus), if it increases to the left a left rotator (left externus, right internus). If the diplopia in either case is homonymous, it is the externus muscle which is paralyzed; if the diplopia is crossed, it is the internus muscle.

If the diplopia is mainly vertical and this vertical diplopia increases fast in looking up, the paralysis affects an elevator. If, then, the vertical diplopia increases

TABLE 2.—*Lagging and Overshoot Produced by Paralysis and Spasm of the Individual Muscles*

Direction of the Gaze	Indications Afforded by Comitance and Screen Comitance Tests: In all cases of paralysis seated in the R. or spasm seated in the L. eye, when the eyes are turned in the given diagnostic direction, the L. eye overshoots when the R. fixes and to a less extent (often much less) the R. eye lags when the L. fixes. In a paralysis seated in the L. or a spasm in the R. eye, the R. overshoots and the L. lags when the other eye fixes		Eye Showing the Greater Deviation	Indicates	
	R. Fixing	L. Fixing		Either Paralysis of	Or Spasm of
Eu & r	R. lags or n.;* L. overshoots	R. lags; L. n.	L	R. sup. rect.	L. inf. obl.
	L. lags; R. n.	R. lags or n.; R. overshoots	R	L. inf. obl.	R. sup. rect.
Eu & l	R. lags or n.; L. overshoots	R. lags; L. n.	L	R. inf. obl.	L. sup. rect.
	L. lags; R. n.	L. lags or n.; R. overshoots	R	L. sup. rect.	R. inf. obl.
Er	R. lags; L. overshoots	R. lags; L. n.†	L	R. ext. rect.	L. int. rect.
	L. lags; R. n.	L. lags; R. overshoots†	R	L. int. rect.	R. ext. rect.
El	R. lags; L. overshoots†	R. lags; L. n.	L	R. int. rect.	L. ext. rect.
	L. lags; R. n.†	L. lags; R. overshoots	R	L. ext. rect.	R. int. rect.
Ed & r	R. lags or n.; L. overshoots	R. lags; L. n.†	L	R. inf. rect.	L. sup. obl.
	L. lags; R. n.	L. lags or n.; R. overshoots†	R	L. sup. obl.	R. inf. rect.
Ed & l	R. lags or n.; L. overshoots†	R. lags; L. n.	L	R. sup. obl.	L. inf. rect.
	L. lags; R. n.†	L. lags or n.; R. overshoots			

* Lags or n., because if deviation is not very great, lagging of a fixing eye may be absent or imperceptible.

† In these directions of the gaze the nose soon prevents fixation by the in-turning eye so that no very certain indications are afforded by the tests.

most in looking up and to the right, the muscle paralyzed must be a right-hand elevator (right superior rectus, left inferior oblique); if it increases up and to the left, the muscle paralyzed is a left-hand elevator (left superior rectus, right inferior oblique). In either case one can tell which eye and therefore which muscle is affected by remembering that the higher image belongs to the paralyzed eye (e. g., the right-eye image is higher in paralysis of the right superior rectus and right inferior oblique muscles).

If the diplopia is mainly vertical and this vertical diplopia increases fast in looking down, the paralysis affects a depressor. If, then, the vertical diplopia increases most in looking down and to the right, the muscle paralyzed must be a right-hand depressor (right inferior rectus, left superior oblique); if it increases

most in looking down and to the left, the muscle paralyzed is a left-hand depressor (left inferior rectus, right superior oblique). In either case one can tell the eye and therefore the muscle affected by remembering that the lower image belongs to the paralyzed eye.

In any case to tell the muscle that is paralyzed one has simply to remember that the image formed by the paralyzed eye always lies on the side toward which the diplopia increases (i. e., is the right-hand image when the diplopia increases to the right, the upper image when the diplopia increases upward, etc.).

And a rule to determine the kind of double vision produced by a given paralysis is: the image belonging to the paralyzed eye is displaced in just that direction in which the affected muscle, if intact, would naturally move the eye. Thus, in paralysis of the right superior rectus muscle, which moves the right eye up and to the left and tilts the vertical meridian to the left, the image of the right eye is up (higher), deflected to the left (crossed, diplopia) and tilted to the left.

This rule shows that in paralysis of the subsidiary adductors (superior rectus, inferior rectus), one expects to find a crossed diplopia, and in paralysis of the subsidiary abductors (the two obliques) one will find a homonymous diplopia; also in paralysis of the superior rectus and superior oblique, an in-tilting and in paralysis of the inferior rectus and inferior oblique muscles an out-tilting of the false image. The lateral diplopia, however, may be and often is neutralized by other factors, and the tilting is often but little marked, so that neither is a safe guide in diagnosis. The diagnosis, in fact, must rest solely on the difference in height of the double images and the portion of the field where this difference is most pronounced.

This fact, enunciated and explained very clearly by Mauthner, is frequently overlooked, many insisting that paresis of the superior and inferior recti muscles must produce crossed, and paresis of the oblique muscles homonymous, diplopia. But, apart from the fact that these pareses are often associated with exophoria or esophoria of sufficient amount to neutralize their lateral diplopia, the natural tendency which the visual axes display to diverge when the eyes are directed up and to converge when they are directed down will work to abrogate the homonymous diplopia due to a paresis of the inferior oblique muscle and the crossed diplopia due to a paresis of the inferior rectus muscle. As a matter of fact I have several times found paresis of the inferior rectus muscles associated with homonymous diplopia and paresis of the superior oblique muscles associated with crossed diplopia.

It is not necessary to make the somewhat elaborate analysis just described in order to arrive at the diagnosis by means of the double images and screen deviation. Such a diagnosis can be had at once from table 3, which serves for even complicated paralyses.

In applying this table it is to be noted that the vertical diplopia (DL, DR) caused by an uncomplicated paralysis of an elevator or

depressor increases progressively and fast as the eyes are carried far laterally (from 38° to 40°) beyond the midline. This often, rather than the increase in the vertical direction, is characteristic. Thus when I say that in a paralysis of the right superior rectus muscle the DL increases in Eu & r, I mean that in this corner of the field the diplopia increases both when the eyes are directed up and when they are directed to the right, and often, perhaps usually, the increase to the right is more striking than the increase upward. It has been thought that in the case of paralysis of the superior or the inferior rectus muscle the diplopia should be at its maximum when the affected eye is abducted 23°, but in my experience it increases progressively as the eye is abducted to its usual limit of motion (40° or so).

TABLE 3.—*Diplopia and Deviation in Paralysis*

Deviation and Diplopia Increasing In	Kind of Diplopia	Kind of Deviation	Regularly but Not Invariably Associated with	Deviation and Diplopia Greater When Fixation Is Done by	Indicates	
					Either Paralysis of	Or Spasm of
E u & r	DL	LH	DX and X >> in E u & l	R	R. sup. rect.	L. inf. obl.
	DR	RH	DH and S >> in E u & l	L	L. inf. obl.	R. sup. rect.
E u & l	DL	LH	DH and S >> in E u & r	R	R. inf. obl.	L. sup. rect.
	DR	RH	DX and X >> in E u & r	L	L. sup. rect.	R. inf. obl.
E r	DH*	S	.....	R	R. ext. rect.	L. int. rect.
	DX*	X	.....	L	L. int. rect.	R. ext. rect.
E l	DX*	X	.....	R	R. int. rect.	L. ext. rect.
	DH*	S	.....	L	L. ext. rect.	R. int. rect.
E d & r	DR	RH	DX and X >> in E d & l	R	R. inf. rect.	L. sup. obl.
	DL	LH	DH and S >> in E d & l	L	L. sup. obl.	R. inf. rect.
E d & l	DR	RH	DH and S >> in E d & r	R	R. sup. obl.	L. inf. rect.
	DL	LH	DX and X >> in E d & l	L	L. inf. rect.	R. sup. obl.

* The increase in diplopia and deviation must be marked in order to make the diagnosis certain.

In using table 3 it must be observed that a lateral diplopia increasing to the right or the left does not always mean a paralysis of the external or the internal rectus muscle. For there are other abductors besides these muscles, namely, the obliques, paresis of which may produce a homonymous diplopia varying with the direction of the gaze. Moreover, a homonymous diplopia increasing to the right or the left does not always mean paralysis of an abductor. It may occur with paralysis of an adductor (the internal rectus and the superior and inferior recti muscles), provided this paralysis is associated with a comitant esotropia of considerable degree. Thus suppose that there is a paresis of the right internus muscle such that when the eye is carried 20° inward there would be a crossed deviation and diplopia of 15°, while the deviation and diplopia in the primary position would be only 5°, and nothing at all when the eyes were directed a little way to the right. Suppose further that the condition had developed on top of an esotropia with a deviation of 20°. This deviation of 20°, being present in all directions of the gaze, would neutralize the crossed diplopia due to the paresis, and the patient would then show a homonymous diplopia of

5° in eyes left, 15° in eyes front and 20° in eyes right. This case, which simulates a paresis of the right externus muscles, would be cleared up by ascertaining in what direction the movements of the eyes were limited. A case like this would be unusual, but quite frequent are the instances in which a homonymous diplopia due to paresis of an oblique muscle or a crossed diplopia due to a paresis of the superior or inferior recti muscles is converted into a diplopia of the opposite kind by the presence of a compensating esotropia or exotropia. It is thus seen that a homonymous diplopia that increases to the right may be caused by paralysis of any abductor (the external rectus or an oblique muscle) of the right eye and also by paralysis of any adductor (the internal, superior or inferior rectus muscle) of the right eye, provided there is a condition present causing a comitant homonymous diplopia which is more than sufficient to equalize the crossed diplopia that the adductor paralysis would cause. Similarly, crossed diplopia increasing to the one side may be caused by paralysis of any of the muscles of one eye.

An analysis of all the possible conditions giving rise to a lateral diplopia leads to the following statements, which I enunciated many years ago:[5]

1. *A homonymous diplopia, whether large or small, which increases as the eyes are carried to the right, indicates underaction of some muscle of the right eye or overaction of some muscle of the left.*

2. *A crossed diplopia which increases as the eyes are carried to the right indicates underaction of some muscle of the left eye or overaction of some muscle of the right eye.*

If the tests for diplopia are made under special conditions, e. g., in such a way that the double images are projected against a horizontal background, these images, instead of appearing on different levels, may appear one behind the other, or a vertical diplopia may even be reversed.

Accessory Means of Diagnosis; Limitation of Monocular Movement.—The means already detailed usually serve to make the diagnosis in even the most complex cases. Only occasionally does one need to use any confirmatory tests. These, as already indicated, are:

1. The range of monocular movement.
2. The pointing test.
3. The torsion tests.

The limitation or excess of excursion of either eye in monocular movement occasionally furnishes a diagnostic clue, especially when the eyes are hampered in their movements by a strong spasm of convergence. Particularly in cases in which there is paralysis of the external rectus muscle and yet one suspects that the muscle has more power than it shows, the ability of the eye to rotate out will often be demonstrated if the other eye is tightly covered. This maneuver is of particular

5. Duane, Alexander: New Classifications of Motor Anomalies of the Eye, 1906.

advantage in the case of young children or of hysterical subjects who may refuse to move the eye beyond the midline when both eyes are open, but will move it out sometimes quite well if the other eye is closed. In general, however, the test of the monocular rotation does not afford nearly as convincing or as satisfactory proof of paralysis as the binocular tests. This is particularly apparent in the case of a moderate paralysis of an elevator and a depressor.

Case 5 is a marked instance of this.

Case 5.—*Paralysis of Both Depressors Right Eye; Monocular Field of Fixation Apparently Normal.*—Mrs. L. H. W., aged 37, had several severe attacks of articular rheumatism at 7, 14 and 27 years of age. In one such attack seven years prior to examination, which was particularly severe (affecting the arms and knees), blurring of vision suddenly developed which compelled her to go about with an eye closed. Since then, especially when using the typewriter, she had attacks of blurred vision, relieved by closing either eye or by tilting the head forward. Sometimes she had marked vertical diplopia.

Vision was 15/15 in both eyes; there were slight hyperopia and astigmatism. The patient held the head tilted to the left shoulder RH (Sc) moderate in EF, << to 0 in El, >> in Ed and marked in both Ed and 1 and Ed and r; DR 7°; at 4 feet, DR of 3 inches in EF >> in ED & r and ED & l, to from 9 to 12 inches, << though still present in Eu, except in Eu and r where she soon had binocular single vision. There was some lagging of the right eye when both eyes were directed down. The monocular rotations, tested on the perimeter, showed:[6]

	Right Eye	Left Eye
Er	68	(60)
Eu & r	68	58
Eu	58	50
Eu & l	60	62
El	(50)	60
Ed & l	(65)	80- 85
Ed & r	75- 80	(50)

The binocular tests pointed clearly to a marked insufficiency of both right depressors, and especially the superior oblique muscle. One would expect, then, a distinct limitation of the movement of the right eye in looking down. But the movement, though apparently a little less than that of the left eye, was fully normal, and the total range of excursion up and down of the right eye was actually a little more than that of the left. It might be argued that the case was one of spasm of the elevators in the left eye, so that when the attempt was made to move the eyes down this eye stayed up. But this view is hardly tenable, since in a case of spasm there would have been a marked upshoot of the right eye in looking up, and the diplopia would have been more marked in the upper field than in the lower field.

Case 6.—*Paralysis of the Left Superior Oblique Muscle with Slight Paresis of Some Branches of the Left Third Nerve.*—In Mrs. H. R., aged 44, three weeks before examination, diplopia developed suddenly and without preliminary headache. She had vertigo, which abated and soon disappeared.

6. The figures enclosed in parentheses denote that further rotation was possible, but the object of fixation was hidden by the nose.

DH and DL were present throughout the lower field. Examination showed DL >> in Ed & r to 10 inches at 3 feet, << in Ed & 1 to nearly 0; DH >> in Ed & 1; in EL, DL & H; in Eu & 1 (far to the left) DL 3 inches and DH. Elsewhere there was binocular single vision. There was no tilting of either double image, and no torsion was shown by the Maddox test. The excursions of the eyes appeared normal, and the left eye in particular showed little or no impairment of movement even in Ed & r. But when both eyes were tested together, the left lagged behind the right in Ed. There were RH and S (Sc), especially in Ed. There was very little deviation (Sc) in the upper field. The right pupil measured 5 mm., the left, 7 mm.; both reacted moderately to light and strongly to convergence. Accommodation was apparently equal in the two eyes. Later there was slight ptosis of the left eye.

The Pointing or Projection Test.—The pointing test is preferably made with each eye separately, the other being closed. A patient looking with the affected eye and trying to direct his finger at an object lying in the field of action of the paralyzed muscle will overshoot the mark. Similarly, if one of his muscles is overacting and he tries to point at an object situated on the field of action of this muscle, he will undershoot.

The pointing test is occasionally of help in difficult cases, especially in those of recent paralysis. Yet it is often impossible to demonstrate false projection by it even in well marked cases of paralysis. Hence it is not proper to say, as some have done, that the absence of false projection is a proof that there is no paralysis present.

Case 7 seemed to indicate this diagnosis.

CASE 7.—*Exotropia Probably Developing from Paralysis of the Right Internal Rectus Muscle with Consecutive Contracture of the Right External Rectus Muscle.*—L. G. A., aged 26, had gradually developed diplopia for two years. He experienced vertigo if he tried to walk with the left eye shut.

Examination showed: X (Sc) 16-17°, X′ 18° or more. Primary and secondary deviation were equal. The excursions of the eyes were normal in all directions and apparently comitant. DX was about the same in all parts of the field; RH, 1-2°. He tilted the head to the right. With the pointing test, the projection of the left eye was normal; the right eye overshot in El and undershot in Er (i. e., in either direction it pointed to the left of the object). This false projection was also apparent by his missing the mark when he tried to pick up anything when the left eye was closed.

The false projection here would be explained on the supposition that there was a primitive paralysis (in this case probably syphilitic) of the right internal rectus muscle followed by contracture and overaction of the right external rectus muscle. The resulting picture was ultimately that of a comitant exotropia. But the evidence of a residual weakness in the right internal rectus muscle and of continuing overaction in the right external rectus muscle was afforded by the pointing test.

When fixation is habitually performed by the paretic eye, projection may be normal in this eye and abnormal in the sound one. This was

seen in two cases reported by Alfred Graefe,[7] as also in a case that I saw of paralysis of the left superior rectus muscle and left abducens nerve, in which the patient habitually fixed with the left eye, and objects appeared steady when viewed with the left eye but showed false motion when viewed with the right eye.

Torsion Tests.—These indicate whether the image formed by one or the other eye is tilted and whether it is tilted to the right or to the left.

As has been said, tilting of the double images may be determined by taking the patient's judgment as to the relative position of the two images of a candle; or, better, may be determined and also measured with the Maddox rod, the amblyoscope or the clinoscope, which will also surely show in which eye the tilting takes place if the test is made with each eye separately. This is not always correctly indicated by

TABLE 4.—*Tilting of Images in Paralysis and Spasm*

Direction of Gaze in Which the Tilting Is Most Marked	A. Image Formed by Right Eye if Tilted to	
	Right Denotes	Left Denotes
Eu and r	Paralysis of R. inferior oblique	Spasm of R. inferior oblique
Eu and l	Spasm of R. superior rectus	Paralysis of R. superior rectus
Ed and r	Spasm of R. superior oblique	Paralysis of R. superior oblique
Ed and l	Paralysis of R. inferior rectus	Spasm of R. inferior rectus
	B. Image Formed by Left Eye if Tilted to	
	Right Denotes	Left Denotes
Eu and r	Paralysis of L. superior rectus	Spasm of L. superior rectus
Eu and l	Spasm of L. inferior oblique	Paralysis of L. inferior oblique
Ed and r	Spasm of L. inferior rectus	Paralysis of L. inferior rectus
Ed and l	Paralysis of L. superior oblique	Spasm of L. superior oblique

the candle test, which, on the other hand, may prove the existence of a torsion that the monocular tests fail to reveal.

The facts with regard to the tilting of the double images in paralysis and spasm may be stated in tabular form (table 4).

Too much stress must not be laid on such a statement. In many cases of paralysis and spasm it is difficult or impossible to demonstrate any tilting, and when present it may not be according to rule. In most cases one does not have to consider the tilting at all in making the diagnosis, which can be established readily by usual methods. The chief place in which a consideration of the tilting comes in is making a diagnosis in the occasional obscure cases in which it is difficult to tell whether one is dealing with a paresis of one muscle or a spasm of its associate. For example, DL >> in Ed & r might in a given case be attributable either to paresis of the left superior oblique muscle or to overaction (contracture) of the right inferior rectus muscle. If it were the former, one would look for a right tilting of the left-eye image, occurring especially when the eyes were directed down and to the left; if it were a contracture of the right inferior rectus muscle, one

7. Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1910, chap. 11, p. 88.

would expect a left tilting of the right-eye image, which should be also most marked when the eyes were directed down and to the left.

Diagnosis in Usual and Unusual Cases.—In the ordinary case the diagnosis presents little difficulty. A paralysis of a lateral rotator—the external or internal rectus muscle—is usually made out at once by the obvious limitation of the outward or inward movement of the affected eye. And for the elevators and depressors the diagnosis is equally easy if one keeps in mind the aphorism laid down to students of the subject: "Look out for the corners." For each of the four corners of the field—the up and right, up and left, down and right and down and left directions of the gaze—has belonging to it its special pair of associates. If, then, any muscle is acting insufficiently or too much, the results will make themselves evident first and chiefly in the corner where that muscle belongs. The experienced observer thus soon gets instinctively to associate each muscle with its corner, and where evidences of trouble develop in any single corner this at once indicates that one of the muscles is affected. Almost mechanically he associates the right superior rectus and the left inferior oblique muscle with the right upper corner and the right superior oblique and the left inferior rectus muscle with the left lower corner. He who in this way memorizes the fields of action of the muscles as already given will rarely be at a loss in his diagnosis.

Diagnosis in Cases of Slight Paralysis.—This statement holds for the cases of moderate paralysis. In these the restriction of movement, the deviation and the diplopia do not appear in any characteristic way in the primary position or near it. But when the eyes are pushed to the limit of their movement in some one of the six diagnostic directions, these symptoms develop in the regular fashion. This is seen particularly in paralyses of the superior and inferior recti muscles. In the extreme corner of the field, a characteristic, though slight, vertical diplopia reveals the presence of a weakness of the corresponding muscle. To make such a diagnosis certain, one must assure oneself by repeated examinations at fair intervals that the characteristic diplopia is constantly present. An inconstant, fluctuating, slight vertical diplopia occurring in the outlying portions of the field is probably in most cases due not to paralysis but to spasm. That is, the patient, in endeavoring to make an extreme movement or to overcome a lateral diplopia, makes irregular spasmodic efforts which throw one of the eyes off level. In a true paralysis, the diagnosis is of importance, since, slight as they are, these insufficiencies seem in some cases, especially when the paralysis affects the inferior rectus muscle, to give persistent trouble.[8] For this

8. In cases like those which have been regarded by some as instances of "physiologic" diplopia, i. e., of diplopia that is thought to occur in the periphery of the field of fixation even in normal persons.

reason it is important, as before intimated, to carry the tests of diplopia and excursion to the limits of the field of fixation.

Diagnosis in Complex Cases.—Just as the principles laid down serve for the diagnosis of simple cases, even when very slight, so also they answer for the mixed and complex cases. The discriminations here are made most readily perhaps by means of the accompanying tables. Thus, if in a given case one finds DL and RH $>>$ in Eu & r; $<<$ to 0 as the midline is approached and changing to DR and RH $>>$ in Eu & l, there is a paralysis of both superior recti muscles. So also, if there is DR in Eu & l changing to a DL in Ed & l, there is a paresis of the left superior and inferior recti muscles. A combination like DR in both Eu and r and Ed & r, but rather more marked in the upper field, is regularly due to a paresis of the right superior muscle with a consecutive contracture of the right inferior rectus muscle.

The diagnosis becomes specially difficult when through consecutive changes the deviation and diplopia become nearly comitant. But even then, as has been already stated, there is usually some characteristic increase of the deviation, constantly occurring in a given direction, which discloses the muscular insufficiencies that were the primary cause of the condition.

Complexities Caused by Spasm of the Associate or Antagonist Muscles.—Confusion in the diagnosis may be occasioned by the fact that the effects of paresis of a muscle are augmented by spasm, often varying, in the antagonist. The picture presented is still more confused when, as often happens, the patient fixes with the paretic eye and thereby exchanges a moderate deviation in this eye for a considerable and opposite deviation in the other. In many cases again, especially of congenital deviation, a fairly considerable disparity in the movements of the two eyes exists, and yet the lagging eye, when examined by itself, moves with considerable freedom, so that it would seem as if the disparity was due not so much to paresis of a muscle in this eye as to the excessive action of the associate in the other. Here, too, therefore, the diagnosis may be at times obscure, in that one cannot say which one of the two associates in question is predominantly at fault. Nevertheless one can always say which predominates in action over the other, and this is generally all that one needs to know for the purpose of treatment. In cases of this sort in which the restriction in movement in one eye is not very great it makes little difference whether one muscle is absolutely too strong or the other absolutely too weak, so long as one knows which is the stronger of the two.

SURGICAL TREATMENT OF CONCOMITANT SQUINT

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In discussing the operative treatment of squint, I shall limit my remarks to concomitant squint because I feel that this type is most frequently neglected. Much more can be accomplished for the young squinter than is being done. The child learns to walk, to talk and to fuse the two retinal images during the first years of life. This, then, is the only time when one can *cure* squint. Could one eliminate the many time-worn fallacies, such as the belief in ultimate cure by the prolonged use of glasses and the vague hope that the child may outgrow the condition, and attack the problem early, from both a scientific and a surgical standpoint, the final results would be far more gratifying.

If glasses do not cure squint, or at least markedly improve it, in from two to three months, they probably never will. Notwithstanding this fact, a majority of young squinters are deluded with this false hope for years, at the price of great mental stress, abnormal and deficient environmental adjustment, much economic loss and, in the end, the tragic loss of a serviceable eye. The very few patients who appear to recover from cross-eye as they grow older do so at the expense of the loss of that eye, for they seldom, if ever, have vision in the previously deviated eye. A blind eye—and these are essentially blind—has a marked tendency to deviate outward. This can explain many of the so-called cures.

I do not wish to underestimate the value of properly fitted glasses or of the various methods of training used in the cure of squint. They are of undoubted value and, when employed early, will cure from 20 to 40 per cent of patients better than surgical intervention. My plea is that when it becomes evident that these methods are not efficacious—and this can be determined in a few months at the most—it is one's duty to set the eye so nearly straight by operation that glasses and fusion training can perfect a cure. When glasses, cycloplegics and orthoptic training cease to reduce the degree of deviation, it becomes an imperative duty to operate while the child is young enough to acquire binocular vision. After this one should renew the efforts to secure fusion. Ophthalmologists have neglected these patients, and this is my reason for presenting operative indications and procedures at this congress. In doing so I shall describe a definite plan of procedure which I have found highly successful.

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Since fusion cannot be acquired except during the first few years of life, it becomes necessary, when other methods fail, to operate while the child is very young. This, in turn, means that one must operate under ether anesthesia. I am aware that under ether the degree of squint is always altered and at times the squint is entirely eliminated; therefore, one must operate on the basis of information gained before the child is placed under an anesthetic. The great importance of preoperative study thus becomes evident. Consequently there must be: first, an accurate preoperative diagnosis; second, accurate preoperative measurements, and third, accurate operative procedures.

DIAGNOSIS

The diagnosis must comprehend a searching study of the etiology of the squint in each case. One must determine whether it is paralytic (wholly or partially) or concomitant, and if paralytic, which muscle or muscles are involved. Owing to the difference in treatment, the paralytic cases are omitted from further discussion in this paper. There are also a certain number of cases due to some pathologic condition, either congenital or acquired in early life. These must be distinguished from the ordinary concomitant squint, as neither the time for operation nor the character of the operative procedure is the same.

In a very high percentage of cases of concomitant squint no perceptible pathologic condition of the fundus can be demonstrated either by means of the ophthalmoscope or by studies of the visual fields. In recent cases, there appears to be little if any change in the relative normal strength of any individual muscle. It is admitted that after a considerable time (which is just what one wishes to avoid) there are often both overdevelopment of the overacting muscle and attenuation of the weaker. When these conditions are present one finds convergence excess and divergence insufficiency (Duane). I have seldom seen convergence insufficiency in convergent squint, although it is frequently found in divergent squint.

Vertical squint of high degree is usually congenital and paralytic. The examination of these eyes in the cardinal directions will usually elicit this fact.

MEASUREMENTS

Accurate measurements are almost as important as accurate operative procedures. Without the records of definite preoperative measurements one is at a loss the moment the patient is placed under ether. One is as helpless as a ship without a rudder. I feel certain that this is the chief reason that men with extensive operative experience are so often heard to say that no one can estimate even approximately what effect can be obtained from a certain number of millimeters of shortening or

recession. If one does not first measure accurately the degree of deviation present and is as inaccurate in the operative procedure, it is not to be wondered at that definite results are not obtained.

For measurements on the very young child, I have found the Priestly-Smith tape efficacious. The Holzer modification of this tape, as recommended by me,[1] used with a concave mirror which focuses about 40 cm., is accurate within 2 or 3 degrees. The measurement is taken at least five times, under different circumstances. An average of these measurements is taken and is sent to the operating room with the patient. The measurements are taken for 60 cm. and for 4 or more meters in order to determine the convergence excess. A tropometric reading is taken to determine whether there is any undue alteration in the action of the individual muscle. An overacting or an underacting muscle must receive special attention. This topic I shall take up under operative procedure.

For patients who will cooperate, one can use the cover or the parallax test, or both, which are without doubt the most accurate methods of measuring the squint. These methods have also the advantage that both the vertical and the horizontal deviation can be accurately measured. The objections are that they are not applicable to a large number of spoiled young children and that they are useless when amblyopia is present.

OPERATION

As ophthalmologists have been careless in the preoperative diagnosis and measurements, so have they been careless as to exactness in surgical intervention; and for this reason alone, the number of operative procedures is ever increasing. I shall endeavor not to present anything radically new, but to see if I cannot, in a measure, emphasize some of the good points in the methods that have been presented and at the same time offer procedures which are simple, safe and efficient.

Primarily, the object of early operation is to restore binocular vision at an age when it can still be secured; at the same time, one must not interfere with the normal relations of convergence and accommodation. The ultimate use of these eyes must be considered and that operative procedure practiced which will as nearly as possible restore to each muscle its normal function. One must not make too large an advancement or shortening and thus inhibit the action of the antagonistic muscle; neither must one so weaken any one muscle that its normal function will be impaired. This is particularly true of the internal rectus muscles.

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1. Wilkinson, O.: Strabismus, Its Etiology and Treatment, St. Louis, C. V. Mosby Company, 1927.

I am fearful that the tendency at present is to cause excessive weakening of the internal rectus muscle by too liberal recession. While I admit that the cosmetic results leave but little to be desired, one must not forget that in all close work—and most work is of this kind—there is need of liberal convergence excess. It has been my observation that the relative action between the external and the internal rectus muscles has been materially altered when liberal recessions have been done on both internal recti. A far better procedure would be to strengthen the weaker external rectus and weaken the overacting internal rectus, thereby dividing the efforts between these antagonistic muscles, always allowing the internal rectus to maintain its normal dominance. By this means one can far better restore the normal relation between these two muscles and at the same time correct the deviation. With proper care an approximation to both normal convergence and divergence can be secured. In amblyopic eyes and in eyes of older persons in which fusion is not to be expected, the more liberal recession and resection can be performed; however, it is unwise to endeavor to secure too much effect by operating on an individual muscle. The object is not to cause one muscle to become overactive or to weaken it so that it becomes underactive.

OPERATIVE INDICATIONS

Age at Which to Operate.—As soon as it can be determined in a given case of simple concomitant squint that glasses, orthoptic training or the use of atropine is not going to correct the deviation, one should operate. This can be determined certainly in a few months. If all children were properly operated on within the first year after the beginning of the deviation, the proportion of real cures would be raised 30 per cent and the number of cases of monolateral blindness would be decreased in like proportion. The majority of cases of squint occur in the second and third years of life, and it seems unscientific to permit these patients to go on from year to year with no corrective aid except a pair of useless glasses, while vicious muscular and nervous habits are formed and in some cases marked amblyopia develops. It is one's duty to operate before these habits have developed. Consequently, I advise that when it is found necessary an operation be performed during the first year of the squint. In cases which develop during the first year of life, it is advisable to wait until the end of the second year.

On Which Muscle to Operate.—One should operate on the strong muscle when it can be determined that it is really overacting, in order to lessen its action. Likewise, one should operate on the weak muscle when it is underacting, in order to increase its strength. The trend at present is to operate too frequently and too freely on the strong internal rectus muscle. In other words, one is making the strong muscle too weak instead of making the weak muscle strong, and in so doing one is

seriously interfering with the ability to do prolonged work. Excessive convergence does not necessarily mean an unusually strong internal rectus muscle; it may mean an excessive nervous stimulus for convergence or an effort to eliminate an inferior or a dissimilar retinal image. In most early cases of squint, it is impossible to discover either weakness or undue strength in any of the ocular muscles by the means now available. The relative strength of all the muscles is usually well within normal; hence, it seems inadvisable to interfere seriously with the power of convergence by performing a double recession, as is practiced and recommended by many.[2] Would it not be more logical to recede the strong muscle less and resect or advance the weaker? For the very young subject with large eyes having a moderate amount of deviation, preference should certainly be given to some form of shortening of one or both external rectus muscles, with or without the use of the brace as described by me.[3]

In divergent squint with convergent insufficiency, it is advisable to advance one or both of the internal rectus muscles and perhaps recede one or both of the external recti. In divergent squint with good convergence and an excess of divergence, tenotomy or liberal recession of one or both of the external recti is indicated.

OPERATIVE PROCEDURES

The operative procedures which I wish to commend consist of resections and recessions. Some form of strengthening of the weaker internal rectus muscle is indicated for the young squinter when there is hope for any degree of binocular vision. Tucking operations are efficient when the degree of squint is slight. Advancement operations are indicated when a marked effect is to be obtained, but this is not recommended. The resection operation which I present is easily measured and easily performed, can be accomplished with a minimum of trauma and danger and at the same time assures the maximum amount of strength and security. In operating on a very young child under an anesthetic, each millimeter of shortening must be measured with the utmost accuracy, and this operative procedure lends itself to accurate measurements.

All shortenings or recessions are accurately measured by calipers. In recession operations the position of the scleral stitch is measured and marked in order that accurate placing of the stitch can be accomplished. After the stitch is placed, its position with reference to the stump is noted and recorded. By this means alone can one eventually estimate

2. Jameson, P. C.: The Surgical Entity of Muscle Recession, Arch. Ophth. **6**:329 (Sept.) 1931. Berens, Conrad: Resection Operation for Strabismus, Arch. Ophth. **8**:321 (Sept.) 1932.

3. Wilkinson,[1] p. 198.

correctly what can be accomplished by a given number of millimeters of recession. This, of course, is predicated on accurate preoperative measurements and accurate postoperative measurements.

In the resection operation, I use a central mattress suture similar to that described by Snellen in 1869, Ewing in 1915 and Lancaster in 1918. This is brought out through the capsule and conjunctiva, as suggested by Duverger in 1919. This mattress suture secures a much stronger anchorage than is obtained by the Reese operation, the resection operation most popular in America.

This resection operation is performed as follows: An incision about 1 cm. long is made in the conjunctiva about 2 mm. in front of the insertion of the muscle. The muscle is exposed approximately 1 cm. through this incision, and the tendon of the muscle lifted on a strabismus hook. A Prince forceps is placed on the muscle about 1 mm. from the stump. The muscle is cut 1 mm. from its insertion and turned back

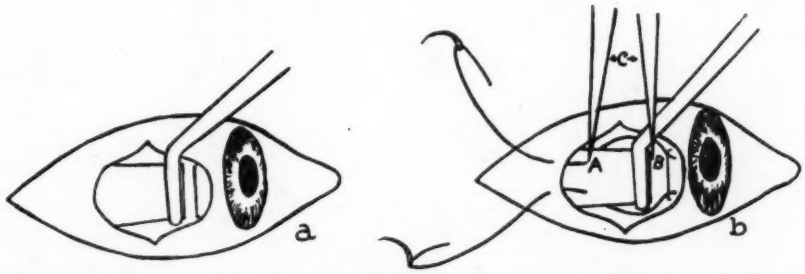

Fig. 1.—In *a*, the external rectus muscle is exposed, clamped with forceps and cut 1 mm. from the insertion. In *b*, the sutures are introduced from within out through the upper and lower fourths of the stump, securing a good scleral hold. They are then brought through the muscle from its under side at a measured distance from the end *B* and then out through the capsule and conjunctiva.

toward the canthus with the forceps. With a short, full-curved needle, a double-armed no. 2 braided silk suture is inserted from the bulbar side, through the upper and lower fourths of the stump, care being taken to secure a good scleral hold (fig. 1 *b*). The scleral stitch is easily introduced and provides a sure anchor for the advanced muscle. The two threads are now passed through the muscle from its under side at a measured distance from the cut end (*A, B* in figure 1 *b*) and then out through the conjunctiva. The eye is pushed over by the assistant, and the muscle brought forward with the Prince forceps still holding it and the thread tied, thus fixing the advanced muscle to its stump with a mattress suture (fig. 2 *a*). The lateral sutures are now placed, one above and one below, passing through the conjunctiva, capsule, muscle and conjunctiva, thereby closing the wound (*E, F* in figure 2 *b*). The amount of shortening to be produced in a given case is determined by

the degree of the deviation that one wishes to correct. Approximately 15 degrees of deviation will be obtained from 10 mm. of shortening. The exact amount of the shortening produced is determined by measuring the distance from the thread at *C* to the cut end of the muscle at *D* (fig. 2*a*).

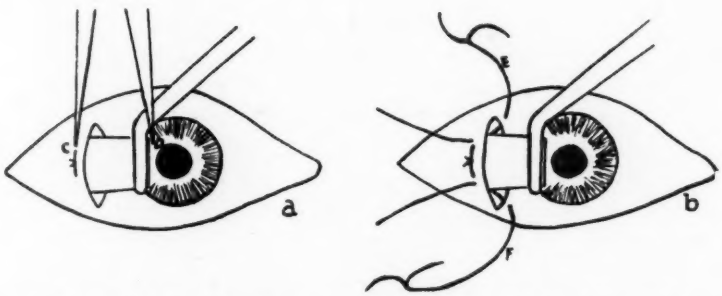

Fig. 2.—In *a*, the eye is pulled up straight, the end of the muscle brought up to or overlapping the corneal margin and the suture tied. *C* and *D* represent the points of the caliper measuring the exact amount of shortening procured. In *b*, the muscle is advanced, the main suture is tied and two lateral sutures are placed. The muscle is cut back of the forceps, and the lateral sutures are tied, thus closing the conjunctival wound. Additional conjunctival sutures are placed if it is found to be necessary.

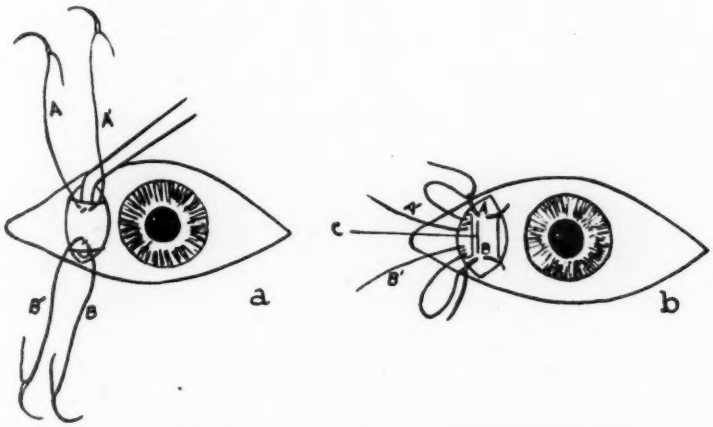

Fig. 3.—In *a*, the tendon is lifted with a hook, and double-armed sutures are whipped into its upper and lower borders. In *b*, the tendon is cut about 1 mm. from its insertion. A mark 6 mm. long is made on the sclera with any pointed instrument from 4 to 5 mm. back of the stump and directly opposite it (*C*). At each end of this scleral mark sutures *A* and *B* are introduced into the sclera.

In cases in which only moderate resections are necessary, Prince's forceps is placed at a measured distance from the insertion of the muscle and the stitches are placed behind the forceps, the position of Prince's forceps on the muscle thus being used as the exact measure of the amount of shortening.

The objection expressed by many to recession is the scleral stitch. However, with practice this can be introduced with relatively little danger. Five millimeters of recession will give about 15 degrees of correction. I usually recede the muscle about 4 or 4.5 mm. and expect to correct about 12 degrees of deviation, particularly if a resection is performed on the opposite muscle. I do not use the lateral cuts into the capsule as recommended by Jameson as I do not wish to produce underaction of the muscle.

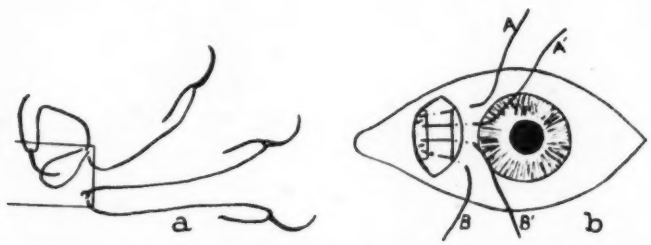

Fig. 4.—In *a* is seen the detail of the lock stitch employed only when chromic catgut is used, showing the passing of the needle through the loop to lock the stitch. In *b*, after sutures *A* and *B* are introduced into the sclera about 8 mm. apart at a marked distance from the stump, they are brought out through the stump and conjunctiva. Sutures *A'* and *B'* are passed through the stump and conjunctiva only.

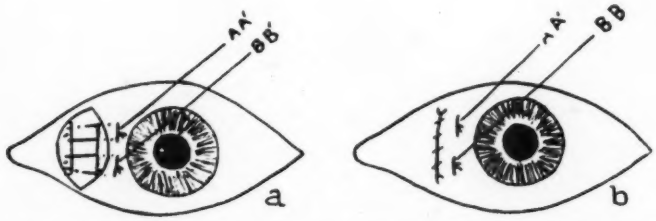

Fig. 5.—In *a*, the end of the muscle is brought up to the scleral anchorage, and the sutures are tied. In *b*, stay sutures are tied, and a running suture closes the conjunctival wound.

I prefer the operation about to be described, as I have found the ultimate scarring less, and I feel that it is as safe and as easy to perform as any recession operation using a scleral stitch.

The recession operation which I wish to present is as follows: The internal rectus muscle is exposed by an incision about 2 mm. anterior to the tendinous insertion and about 1 cm. long. A tendon hook is placed beneath the tendon. Two double-armed twenty day 000 chromic catgut sutures are whipped and locked into the tendon at its upper and lower borders, directly over the tendon hook (fig. 3*a*). For older patients I use no. 5 twisted silk. This is whipped into the borders of the tendon but not locked, as I have found the locked stitch difficult to

remove. The muscle is now cut in front of the sutures, 1 mm. of stump being left. A mark 6 mm. long is made on the sclera about 4 or 5 mm. from the cut stump (*C* in figure 3 *b*). This is made with a pointed instrument and stained with a 1 per cent aqueous solution of acriviolet. At each end of the mark on the sclera, one arm of each pair of threads is introduced into the sclera for about 2 mm. and is then passed through the stump and out through the conjunctiva about 3 mm. from the cornea. The other two arms (*A* and *B* in figure 4 *b*) are placed through

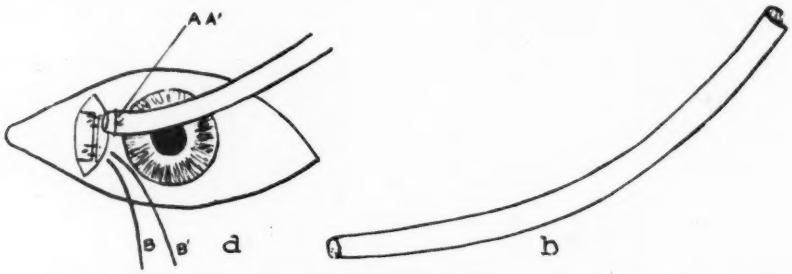

Fig. 6.—In *a*, the muscle is cut, and pairs of sutures are passed through the stump and out through the conjunctiva about 3 mm. from the cornea. The upper sutures are shown drawn up over a rod 4 or 5 mm. in diameter and tied so as to set the muscle back from its stump that exact amount. The lower sutures are yet to be tied in the same manner. In *b* is seen a slightly curved metal rod 6 inches (15 cm.) long, the ends of which are oval, one being 3 mm. by 2.5 mm., increasing to 4 mm. by 3 mm. at a point 5 mm. from the end. The other end is 3 by 5 mm.

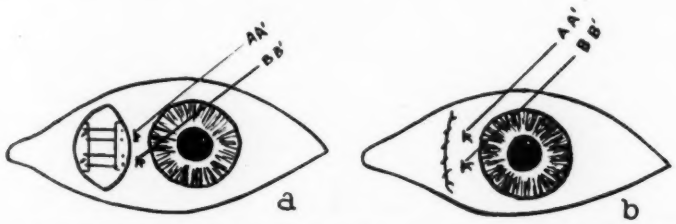

Fig. 7.—In *a*, the slack is taken out of the two stay sutures *AA'* and *BB'*, allowing the muscle to recede 4 or 5 mm. In *b*, the wound is closed tight with a running suture. *AA'* and *BB'* represent the stay sutures.

the stump and out through the conjunctiva. The end of the muscle is drawn up to the scleral anchorage, and the pairs of threads are tied. The running suture closes the conjunctival wound (figures 3, 4 *b* and 5 show the various steps of the operation).

I have recently devised a recession operation without a scleral stitch which I feel is safe and at the same time the most simple recession operation possible. In this operation the tendon of the internal rectus muscle is exposed only enough to permit a tendon hook to be placed under the tendon. Two double-armed sutures are whipped into the

upper and lower borders of the tendon directly over the tendon hook (fig. 3 *a*). The capsule is disturbed as little as possible. The muscle is cut 1 mm. from the stump and held away from the sclera by threads, while the sclera is scarified with a small curet at a point about 4 mm. back of the muscle stump. The pairs of sutures are now brought through the stump at its lower and upper edges respectively and out through the conjunctiva and tied over a 4 or 5 mm. rod (fig. 6 *a*). The rod is withdrawn, and the end of the muscle recedes from 4 to 5 mm. from the stump. If the capsule is unduly opened, lateral sutures uniting the capsule and the cut muscle tendon, as suggested by Mott,[4] are recommended. A continuous suture now closes the conjunctival wound (figs. 3 *a*, 6 and 7). The stay sutures locked into the tendon and brought out through the stump, the scarification of the sclera opposite the cut tendon, with the slightest possible disturbance of the capsule, and the accurate closure of the conjunctival wound assure definite end-results with the best cosmetic effect.

I have performed twenty-five of these operations during the past two years and shall report on them later. My results so far have been good. About 12 degrees of correction can be secured with a 4 or 4.5 mm. recession, and the cosmetic and functional results are excellent.

1408 L Street, N. W.

4. Mott, W. C.: Muscle Recession with Tendon Stump and Tenon's Capsule Fixation, Arch. Ophth. **8**:858 (Dec.) 1932.

SOME POSSIBILITIES OF ORTHOPTIC TRAINING

A COMPARATIVE STUDY OF A CONTROL GROUP AND A TREATED GROUP OF PATIENTS WITH SQUINT, WITH REMARKS CONCERNING THE TECHNIC USED IN ORTHOPTIC TRAINING

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CHICAGO

The status of the orthoptic treatment of squint and the anomalies of muscle balance is not definitely established. Many ophthalmologists regard its results as unreliable and not worthy of the effort involved. Exaggerated reports of results by spectacular instruments in the hands of irregular practitioners have tended to cast discredit on the whole method of procedure.

On the other hand, a number of favorable reports have appeared in recent years by ophthalmologists whose judgment cannot be lightly set aside. Notable among these is Ernest Maddox, whose organization of special groups for orthoptic training in London has stimulated interest in the subject among many ophthalmologists.

Ophthalmologists, however, who are authorities and whose experiences extend over years do not always agree concerning the percentage of recoveries secured by orthoptic treatment, the ages of the patients who are benefited most by orthoptic treatment and the causes of squint, which will not be discussed in this article.

Worth [1] definitely stated that 7 years is the upper age limit for treatment. Children between 3 and 5 years secure the best results. After 6 years it is seldom worth while to attempt fusion training. He also remarked that if constant occlusion fixation is not produced after three weeks the case is hopeless.

Peter [2] agreed with Worth and stated that 50 per cent of cures can be obtained in children under 7 years of age. Sattler [3] agreed that early

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This study was done with the aid of a fellowship supplied by the Knapp Testimonial Fund and a grant from the Academy of Ophthalmology and Otolaryngology.

1. Worth, C. A.: Squint, London, Baillière, Tindall & Cox, 1921, p. 112; Discussion on the Treatment of Manifest Concomitant Squint, Tr. Ophth. Soc. U. Kingdom **41**:119, 1921.

2. Peter: Symposium on the Treatment of Nonparalytic Squint, Tr. Ophth. Soc. U. Kingdom **52**:325, 1932.

3. Sattler, C. H.: Deutsche med. Wchnschr. **54**:771 (May 11) 1928.

treatment yields the best results. Maddox [4] stated that the upper age limit of 7 years has been disproved, that fusion may be developed in adolescence and early adult life and that an absent fusion faculty is rarely found. Hansell and Reber [5] were optimistic in their discussion of orthoptic training. They expressed the belief that vision of 20/80 is necessary for comfortable fusion. They defined amblyopia as vision less than 20/80. No statistics are given, but occlusion and atropinization have not been extremely successful in improving visual acuity in their cases. By orthoptic training they believe that results can be secured in almost all cases of alternating squint, including cases in older patients. Rasquin [6] and Onfray [7] expressed the opinion that in most cases of squint fusion will develop even up to the age of 17, and that orthoptic treatments with the Remy diploscope give better results than surgical intervention alone. The two methods combined produce better results than either alone. Onfray said that only 7 per cent of amblyopias improve, and that if vision is below 20/80, improvement seldom occurs.

Sattler [8] did not agree with Onfray and reported 52 cases without central fixation in which he produced satisfactory vision. He failed to secure binocular vision in only 18 of 200 cases. Sattler used prisms up to 20 degrees for constant wear to stimulate binocular macular perception.

Interesting reports are presented by Krusius,[9] who has corrected squint in six weeks by orthoptic training.

Deloge,[10] in developing visual acuity and fusion, reported several cases in which the vision was decreased by keratitis and by a paracentral leukoma and in which he was able to improve visual acuity from 20/200 to 20/25. He mentioned a 32 degree divergence which was absent after fusion training. This divergence was present in patients over 7 years of age. Deloge recalls a case reported by Javal; the patient, aged 14, had a right convergence of 30 degrees and myopia of 8 diopters, and was relieved by treatments extending over a year. He has seen patients who could fuse with 15 diopters of anisometropia and some whose divergence was relieved after ten orthoptic treatments.

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4. Maddox: Tr. Ophth. Soc. U. Kingdom **51**:296, 1931; **52**:440, 1932.

5. Hansell, H. F., and Reber, Wendell: The Ocular Muscles, Philadelphia, P. Blakiston's Son & Co., 1912, p. 171.

6. Rasquin: Ann. d'ocul. **150**:25, 1913.

7. Onfray, R.: Ann. d'ocul. **157**:727, 1920.

8. Sattler, C. H.: Klin. Monatsbl. f. Augenh. **81**:182 (Aug. 31) 1928; footnote 3.

9. Krusius, F. F.: Arch. f. Augenh. **66**:1, 1910.

10. Deloge, C.: Am. J. Ophth. **4**:407, 1912.

In 1925, Goar [11] reported 2 cases of alternating convergent squint in which excellent stereoscopic fusion developed following operative procedures. This author reported 20 per cent of his cases of convergent squint to be of the alternating type. He mentioned the difficulty in making a diagnosis of alternating strabismus. He also discussed a series of 18 patients with alternating convergent squint who were treated by Javal, in 8 of whom stereoscopic fusion developed, with excellent cosmetic results. During the discussion of these cases, Lancaster, Peter, Wilkinson and Verhoeff discussed the importance of determining binocular fusion. Verhoeff did not believe that the patients in the cases reported had true binocular vision. He does not believe that bar reading is a test of binocular vision. The Hering drop test and cover test he believes to be accurate tests for binocular vision.

Schäfer [12] defined Hering's fall test as being one to show a binocular mechanism for perception independent of movements of the eyes. This test may be passed by patients who are unable to fuse in the stereoscope.

Langlands,[13] in reviewing his experiences, expressed the belief that inexperienced observers who fail with the stereoscope sometimes pass a test for stereoscopic ability with the Howard apparatus.[14]

Coulter [15] reported the cases of 5 patients, 1 over 18 years of age, in whom stereoscopic fusion was developed after operation.

Hesse [16] and Aubineau [17] discussed their results in detail. Hesse reported complete recovery in 68 per cent. Aubineau reported recovery in 37.5 per cent of cases of convergent squint and in 13.25 per cent of cases of divergent squint.

Sattler's [8] charts are described in several articles in the German literature. These charts are split and are numbered from 1-1*A* to 45-45*A*. There are, therefore, forty-five split charts. All of them show stereoscopic depth except charts 1 and 2, which are used to test for nonstereoscopic fusion ability. These charts are especially valuable in training younger patients. It is impossible for the subject to learn all the charts, which is so easily done with the E series of Wells.[18] Likewise, the pictures in the E series of Wells cannot be alternated.

11. Goar, E. L.: Tr. Sect. Ophth., A. M. A., 1925, p. 50.

12. Schäfer, Shane: Physiology, New York, The Macmillan Company, 1900, vol. 2, p. 1134.

13. Langlands, N. M. S.: Experiments on Binocular Vision, Medical Research Council Special Series, no. 163, London, His Majesty's Stationery Office, 1929, p. 10.

14. Howard: Am. J. Ophth. **2**:656, 1919.

15. Coulter: Ophthalmoscope **5**:625, 1907.

16. Hesse, R.: Arch. f. Ophth. **128**:145, 1932.

17. Aubineau: Ann. d'ocul. **150**:28, 1913.

18. Wells, D. W.: The Stereoscope in Ophthalmology, Boston, Globe Optical Company, 1918, vol. 1, p. 14.

This may be done with the Sattler charts, producing an alternation of stereoscopic effect.

Sattler's charts embody several important principles which one may use to special advantage in early fusion training. There are definite gradations in the difficulty of charts. The numbers on the charts, however, do not correspond with the more difficult pictures. For example, charts 5 and 20 are much more difficult to fuse accurately than charts 25, 44 and 33. The easier charts are those such as chart 22, which include pictures of a tub, one arrangement looking into the tub and the second looking on the bottom of the tub. Those slightly more difficult are charts 9, 21 and 10, which show a person jumping over a chair or a ball. When seen stereoscopically, the person may be closer or farther from one than the ball. The most difficult chart is 20, which shows a spear, the handle of which is away from one and the point toward one; this chart may be changed to produce the opposite effect. Chart 33 contains 2 boys and 2 girls marching and blowing trumpets. There are, therefore, 4 persons and 4 trumpets to compare in the relative position, which is quite difficult at times.

Variation is secured by the charts. Round, square, circular and irregular-shaped objects appear stereoscopically as single objects or combined with each other. One chart, no. 4, has white objects on a black background; this seems more stimulating at times than the other charts, which present black objects on a white background. The charts are especially valuable in training fusion but also for training fusion amplitude. Convergence ability can be trained by varying the distance between the charts in the stereoscope.

The use of the cheiroscope is well explained by Maddox.[19] The definite use of the same arm and same eye, however, is being discarded. Any one who has attempted to make right-handed patients who are not cooperative draw such pictures with the left hand will realize that it sometimes is impossible.

Smith [20] discussed the diploscope; Berens,[21] the use of prisms; Howard [14] the use of the Howard apparatus in the diagnosis of stereoscopic fusion, and Moncreiff,[22] the diagnosis and presence of abnormal correspondence in convergent squint.

Every one is familiar with the old type of stereoscopic pictures. It is most difficult to prove that patients are seeing them stereoscopically. One may examine such pictures and describe stereoscopic details with-

19. Maddox, E. E.: Proc. Roy. Soc. Med. **23**:48 (Nov.) 1929.

20. Smith: Proc. Roy. Soc. Med. **21**:700 (Feb.) 1928.

21. Berens, C.; Losey, R. R., and Stark, E. K.: New York State J. Med. **29**:663, 1929.

22. Moncreiff, W. F.: Disturbances of Visual Functions in Concomitant Convergent Squint, Arch. Ophth. **2**:179 (Aug.) 1929.

out looking through the stereoscope. The checking of fusion by a vertical line before one eye and a horizontal line before the other proves nonstereoscopic fusion but not actual depth perception. For proof of actual stereoscopic ability with these views, such as those furnished by the Keystone Company, I have a box of small colored and uncolored paper stars, crescents, arrows and circles. These figures are pasted one before each eye, the distance between them being varied from 95 to 75 mm. These flat paper objects, when fused, appear in front or in back of the picture. They can be changed in position at will and can be pointed to as a definite check on the patient's stereoscopic ability. The glue does not adhere firmly to the glossy picture, and so they can be easily removed and do not mar the picture. Fusion amplitude training is difficult with these stereoscopic pictures, and so I prefer the Sattler charts for this purpose.

Previous records concerning the results of the different methods used in the treatment of squint are, on the whole, unsatisfactory. For example, the actual effects in percentages of correcting the refractive error alone and also in conjunction with occlusion and atropinization of the fixing eye are not known. Similarly, there are fewer reports of the results secured by orthoptic treatment alone than reports on orthoptic treatment aided by surgical intervention, or by surgical means alone.

It seemed desirable, therefore, to determine in an American clinic just what could be accomplished by orthoptic training in various types of squint and to provide a center where patients from other clinics could be given training under the most favorable conditions. This was undertaken by the department of ophthalmology of the Northwestern University Medical School, with the assistance of a grant from the Knapp Testimonial Fund. Hours were arranged when the clinic was otherwise unoccupied, and a group of cases was referred to me for examination, treatment and the careful recording of the results obtained. This clinical study was begun on Jan. 1, 1932.

During the period from Jan. 1, 1932, to Feb. 27, 1933, 107 patients were seen in the orthoptic clinic at Northwestern University Medical School. Of these, a number were not included in the series herein reported either because they did not show constant squint or because they were too irregular in attendance. This left a total of 48 patients with concomitant squint, who appeared approximately once each week for observation and intensive training with the amblyoscope, stereoscope and synoptophore.

At the suggestion of Dr. Harry Friedenwald and with the cooperation of Dr. Richard Gamble, a control clinic was established at the Children's Memorial Hospital. At this institution no orthoptic treatment has been given in the clinic. The ordinary measures exclusive

of fusion training, such as the correction of the refractive error, occlusion and atropinization of the fixing eye, were used in this series from Sept. 1, 1932, to Feb. 27, 1933. These patients had all worn glasses for six months before other active treatment was given. Ninety patients were seen at the control clinic. Because of irregular attendance, only 40 cases were suitable for inclusion as a control series. These patients appeared once a week for observation and suggested occlusion or atropinization of the fixing eye. At each visit a determination of the patient's binocular fixation, binocular vision and stereoscopic ability was made and recorded by the same methods as those used at the orthoptic clinic of Northwestern University Medical School. If patients bought stereoscopes and attempted orthoptic treatment at home this practice was not discouraged. Such patients are not included in this report.

The procedures and methods used in the orthoptic clinic in securing information preliminary to training are as follows. In each case of squint a systematic history is obtained and an objective and subjective examination of the muscles and fusion is made. This requires from forty minutes to an hour for completion. The patients are usually referred from the general clinic for diseases of the eye, where refraction under atropine is done and the necessary glasses are prescribed.

In the history are noted: (*a*) the age of onset; (*b*) the type of squint (stages, if present); (*c*) the causes, if any; (*d*) previous treatment (occlusion, atropinization, surgical intervention and orthoptic training), and (*e*) diplopia, if ever present.

The examination is subdivided into: (1) vision with and without glasses, (2) muscle balance and (3) degree of fusion, as shown in the succeeding outlines. These outlines show the methods of recording muscle balance and fusion tests.

Examination of Muscle Balance

Motility: of each eye

Fixing power: of each eye in the primary position and in extreme abduction and adduction

Angle of anomaly: without glasses
with glasses

Red glass diplopia: with correction by a prism (ten minutes)

Maddox rod: without glasses—far—near
with glasses—far—near

Examination for Presence or Absence of Binocular Vision and Degree of Fusion

Monocular Vision:

Amblyoscope: After fifteen minutes, unable to see bird and cage simultaneously

Stereoscope: Without prisms and with prisms, unable to see bird and cage simultaneously (after five minutes)

Synoptophore: After five minutes, unable to see Negro, chicken and duck simultaneously.

Binocular Vision:

1. Bird and cage seen but not superimposed or associated.
2. Bird and cage superimposed or associated for short periods only. Certain details in each picture are not seen.
3. Bird and cage superimposed. Bird kept in cage and maintained in position. All details noted. Some amplitude in moving tubes of amblyoscope or synoptophore or cross arm in stereoscope.

Stereoscopic Vision:

Bucket seen inside and out. Ability to associate pictures in E series of Wells' charts and Sattler's charts. Correct depth perception.

Fusion Amplitude:

Ability to maintain stereoscopic vision while overcoming prisms or moving tubes of amblyoscope and synoptophore.

In examining the motility, one eye is covered while its fellow follows the light of the ophthalmoscope. The excursions of each eye are noted, especially the ability to maintain any position toward the extremity of the field of rotation.

The power of fixation is noted with the ophthalmoscopic light during the examination for motility as described. The ability to fix determines whether occlusion is to be employed, immediately or later, to improve monocular visual acuity. Ability to fix with each eye, even though the suppression of one image occurs, is important in that fusion may be developed more quickly in such cases.

Monocular vision with rapid alternation of the eyes (pseudo-binocularism) cannot be present if simultaneous fixation is developed. This rapid alternation of the eyes with monocular vision should be noted as it prolongs orthoptic treatment by simulating, at times, binocular vision and stereoscopic sense.

In measuring the angle of anomaly or the angle of deviation, the perimeter is used. It is adjusted so that the deviating eye is accurately in the center of the arc, and the patient is told to fix with the non-squinting eye on a light 20 feet distant, which is on a line with the squinting eye and the center of the perimeter. A second light, with the eye of the examiner looking exactly over the top of this light, is carried around the arc of the perimeter until the reflection of the second light lies in the center of the cornea of the squinting eye. The location of the second light on the graduated arc of the perimeter gives the angle of anomaly in degrees.

The angle gamma, or the angle kappa of Landolt, is measured by letting the patient fix the center of the arc with the squinting eye (the

other eye being covered). The light of the ophthalmoscope is carried along until its reflection is in the center of the cornea. The position of the light on the graduated arc shows the size of the angle gamma. In convergent squint, for example, if the angle gamma is positive, it is added to the perimetric measurement of the convergence; whereas if the angle is negative, it is subtracted. The opposite holds for divergent squint.

Diplopia with the red glass before the fixing eye is usually difficult but not impossible to produce in cases of squint. The same may be said of the Maddox rod tests. The examiner must be on the watch for monocular vision of alternating type. The patient knows that there should be two lights and so says that he sees them. He does see two lights but not simultaneously. For the sake of brevity this alternation with some association of the visual images may be called pseudo-binocular vision. It should be noted and watched for in all training exercises and in diagnosis of the squint.

Tests for the detection of monocular vision, binocular fixation, binocular fusion and stereoscopic ability are made by using the amblyoscope, the stereoscope, with and without prisms, and the synoptophore. The charts used are those of Worth, Wells, Sattler and Hamblin. In such tests for the absence or presence of the fusion sense it is especially important to know that the patient fixes binocularly and simultaneously: For example, some patients will fix with one eye, then alternate their vision and obtain a fair degree of accuracy (being able to tell time on the clock chart and to place the bird in the cage) and thus stimulate fusion.

Having determined the angle of squint, the amblyoscope is set at the required angle, and the bird and cage are placed in the tubes of the instrument. The larger object should be before the poorer eye. The patient is asked what he sees. In most instances the image or picture of the fixing eye is described. Then the fixing eye is occluded by holding the hand at the end of the tube, and the suppressed eye is stimulated by moving the picture or by increasing the illumination behind the picture. In a few moments the patient is able to describe inaccurately the picture before the suppressed eye. He is asked to place the bird in the cage, to tell time and to place the parts of man and dog together. He may be allowed to move the tubes of the amblyoscope (slowly) to aid him. If binocular vision no. 1 is not determined after ten minutes, efforts to determine fusion power are discontinued.

If the objects can be fused, the picture of the tub is placed in the amblyoscope. The patient is asked if the larger circles are closer or

farther away, or if he is looking on the bottom or the inside of the tub. The cards are removed and without the knowledge of the patient are replaced in the carriers in exactly the same position as they had been at first. This removal is repeated a third time, except that the cards are exchanged. A patient cannot constantly guess correctly if this procedure is followed. If these stereoscopic cards are fused, which seldom happens, the tubes of the amblyoscope are converged and diverged to see how much fusion amplitude exists. Usually there is little or none.

The stereoscope is next given to the patient with certain charts in the holder. Wells' charts *A*1, *A*2, *A*3, *C*1, *C*2, *E*1, *E*2 and *E*3 and Sattler's chart 4 are given the patient. He sees only one chart before looking through the stereoscope. This is the bird and the cage. While the patient's eyes are being watched to see that he fixes simultaneously, he is asked to describe what he sees and to point to the objects seen. Prisms may be used before the better eye to see if fusion is present. By pointing to the details on the cards, the patient begins a practice which is helpful to the physician in the education of his patients with squint. It tells the examiner, in the use of the cheiroscope and the stereoscope, whether or not the patient is fusing by the location of the objects on the cards, even though the patient may describe the cards in some detail.

If the patient points to only one picture as it is on the chart, he has monocular fixation only and is suppressing one image. If he points to both pictures constantly and he is fixing simultaneously, he has binocular vision no. 1 without fusion. If he points and describes the fusion of the two parts of the card, he is fusing with binocular vision no. 2, and if he is able to maintain the fusion when the cross bar of the stereoscope is moved forward and backward, he has binocular vision no. 3. It is essential that the patient should never know how to point to the details as described, or else the accuracy of the check system is destroyed. This pointing is used also in checking fusion ability during the periods of treatment.

At the patient's first visit to the clinic, if he has had no orthoptic training, he does not know the stereoscopic cards (the E series of Wells, for example). It is a relatively easy matter, therefore, to determine his stereoscopic ability. If a patient has had orthoptic training with these charts, he has often learned them. He has also learned the charts in the amblyoscope. All of these he can describe in detail without and with the stereoscope or amblyoscope. Similarly, after several periods of training the patients learn most of the charts. They are so anxious to please and to be complimented that they will describe the charts and point accurately to details even though monocular vision

is present. How can one discover this condition of affairs, especially when its persistence prevents further progress?

Three methods for checking the absence of fusion suggest themselves: First, the patient can be questioned before the charts are inserted in the stereoscope. If he knows the charts as fused, one cannot be certain that he sees them stereoscopically unless one can invert or vary the picture which the patient sees. For this purpose charts *E*3, *E*5 and *E*6 can be cut into halves. Thus in one view one looks into the pans or figures. By exchanging the positions of the half pictures one can be looking on the bottom of the pans. Even so, after a time patients will learn these pictures and can tell what view is presenting without using the stereoscope. One therefore must have other check pictures about which the patient knows nothing. For this purpose charts *B*3, *C*3 and *C*8 are always removed from the set before presenting them to the patients. As a further check, Sattler's charts are used.

In using the synoptophore, slides 1 and 2 are inserted in their slots. These slides present a picture of a Negro boy with a chicken on his right and on his left a duck with a hat on its head. With the synoptophore regulated for his angle of squint, the patient is asked what he sees. The dimmer is used in front of his good or fixing eye, and he then describes the picture before the suppressed eye. After a few minutes he is allowed to view both objects simultaneously. Gradually the idea that one Negro, one duck and a chicken are visible, if properly fused, occurs, and the first stage of fusion is present. In testing for stereoscopic ability, slides 5 and 6 are used. These show fusion amplitude and stereoscopic duction power when the tubes are moved or the pictures are rotated.

In conclusion, it may be said that the most important facts to note in diagnosis are the presence or absence of pseudobinocularism, the degree of squint, fusion ability and vision in the amblyopic eye.

During the course of treatment cases of squint seem to pass through two definite stages: a period of disorganization and one of reorganization. Thus to break up certain habits or associations one must produce disorganization of accommodation convergence associations by the use of atropine, convex lenses, prisms and surgical intervention—in other words, produce diplopia. One notes this when the patient blinks and tilts his head, or covers one eye, and if asked he will often admit seeing double. Objectively, an increase in the angle of anomaly of rapid nystagmus and separate monocular movements similar to those seen in infants before binocular single vision has occurred are noted. Gradually the monocular squint becomes temporarily alternating.

This stage of disorganization is present at the time the surgeon discharges his patient following surgical correction of squint. What

happens from this time on determines the results obtained, that is, if fusion develops spontaneously or by training. Excellent cosmetic results are obtained, the exception occurring in cases in which one eye is so amblyopic that no such stage of disorganization appears. During this stage in amblyopic eyes, vision begins to improve, fixation becomes less dominant in the fixing eye and better in the squinting eye, so that a true alternation occurs for a time. When this temporary alternation is present, the period of reorganization in orthoptic training should begin. This period is subdivided into (1) a fusion stage, (2) fusion amplitude and (3) the stage in which only phoria remains. These three stages, which are really poorly defined in actual practice, are separated for the sake of clearness. They often merge so closely that they cannot be separated.

This brings up the question whether occlusion or atropinization is a preferable adjunct to fusion training. Worth describes the difficulty of using occlusion. If one places oneself in the patient's predicament of an immediate partial or complete incapacity, with a possible or probable improvement of vision in the future, which would one choose? The cooperation of the patient is most difficult to secure. He is unable to play, perhaps bumps into doors or falls. One sees, therefore, that in most cases complete or partial occlusion is seldom practicable at first. It does not produce binocular simultaneous fixation directly. Its main use is to produce an increase in monocular visual acuity, and this is its indication.

Atropine temporarily suspends the power of accommodation of the eye. If an eye is emmetropic or if one has total correction for refractive error, distant objects are seen clearly, whereas near objects are seen poorly. Worth uses atropine in the fixing eye only of those whose vision is not much less than 6/36. He said: "The use of atropine in the fixing eye only is an exceedingly efficient curative measure." Treatment is continued until the two eyes have an equal visual acuity or no improvement is noted in the suppressed eye. Atropine is preferred by many authorities. I have found that atropine can be used without much cooperation from the patient. Its effects are not especially noticeable cosmetically. It stimulates monocular vision and binocular vision. It dissociates the convergence accommodation reflex. Its action is constant, and not for a few hours each day. With all of its advantages, this drug is the method of choice in the clinic. Its one defect is that photophobia occurs in the fixing eye. Its use must, however, be attended by definite indications. For example, it can be used to suppress the fixing eye so that the squinting eye becomes the fixing eye. Unless one wishes to prolong orthoptic treatment, this is considered overatropinization. The indications for the use of

atropine are: (1) monocular fixation with deviation and suppression of the fellow eye, (2) lack of alternating ability, (3) latent tropias, (4) suppression of one eye and (5) disproportion in visual acuity.

If one remembers that the eyes pass through definite stages with atropinization and that the main purpose is first to produce a disruption of the existing faulty balance, which can be trained gradually into the power to fix binocularly and to see binocularly in the stereoscope, one will use this drug to best advantage.

At Northwestern University 3 drops of 1 per cent solution of atropine sulphate is used for three days as the average initial dose. (Rarely has the fixing eye been found to squint following this medication. Alternation is present on the second visit, which is one week later, and diplopia is frequently present. This complete cycloplegia disrupts the accommodation convergence reflex and lowers the visual acuity for near and far [unless the complete refractive error is corrected] in the fixing eye.) The best results are secured when the visual acuity of the fixing eye is made equal or somewhat less than that of the squint eye, by atropinization. Therefore, the visual acuity should be determined at each visit during its use.

The instrument of most value in orthoptic training is the stereoscope. With this one may train fusion and increase fusion amplitude, dissociate convergence and accommodation, training either at will. One may train the abductors or adductors by moving the holder nearer or away as indicated. With prism held in front one may build up power to overcome prisms up to 75 degrees or more. A point to remember in its use is that the + 5.00 spheres are decentered so that an 8 degree prism base out is secured at 20 cm. The distance between the usual axis of the observer's eyes is not a factor when the slide is 20 cm. from the lenses. At a distance of 20 cm., if the eyes are emmetropic and orthophoric, convergence is zero when the distance between the objects in the charts is 9.5 cm. (There is some psychic convergence, but this is small.) Accommodation, too, is zero when the slides are focused at 20 cm. from the lenses. The rules to remember in using this instrument are: (1) Moving the charts away from the observer increases the power of convergence and decreases the power of accommodation, and (2) moving the slides toward the observer increases the power of accommodation and decreases the demand for convergence. Fusion amplitude can, therefore, be measured and trained by the stereoscope, with and without prisms, by moving the charts. Duction exercises likewise can be performed; the prisms are used base out and the charts are moved away from the observer for adduction; the prisms are used base in and the charts are moved toward the observer for abduction. This last procedure is rarely necessary.

In anomalies of divergence in which fusion is good but no amplitude is present, the stereoscope is invaluable in training the power of convergence. The patients, if using prisms alone, unconsciously suppress, but with the stereoscope they become conscious when stereoscopic vision is present, and also when one image is being suppressed. In cases of squint of 20 degrees or less, the stereoscope can many times be used with prisms, the previous amblyoscopic exercises being omitted.

RESULTS

The number of cases in each group is not large but is large enough to give one an idea as to what therapeutic results each method of procedure will produce in the handling of cases of squint. In the 40 control cases at the Children's Memorial Hospital, atropinization and occlusion of the squinting eye were done without orthoptic training. The indication for occlusion was to improve the visual acuity of the

TABLE 1.—*Series I: Diagnosis and Age Groups; Forty Cases (Controls)*

Ages	Monocular Convergent Squint	Alternating Convergent Squint	Monocular Divergent Squint	Alternating Divergent Squint
2- 4	13	5	..	..
5- 7	9	2	..	..
8-12	5	..	3	3
13-21	0	..	..	..
Totals	27	7	3	3

squinting eye. The indications for atropinization were lack of alternating ability, disproportion in visual acuity, suppression of one eye and the presence of latent tropias or phorias persisting after the angle of anomaly was absent.

As each patient appeared once a week, the angle of squint with and without glasses was recorded. The ability to alternate and to fix binocularly was noted. The patient's ability to fuse was tested with the amblyoscope; the pointing test and check cards in the stereoscope were also used. Even though an eye was squinting, if it was used as the fixing eye in the stereoscope, it was atropinized.

Of these 40 control cases (table 1), 85 per cent were of convergent squint, 17.5 per cent of these being alternating and 67.5 per cent monocular; 7.5 per cent were of alternating divergent squint, and 7.5 per cent were of monocular divergent squint. The ages of the patients ranged from 2 to 13 years (table 1). In the groups below 7 years, 72.5 per cent had convergent squint, 55 per cent of which was monocular and 17.5 per cent alternating. The remaining 27.5 per cent were in the 8 to 12 year group. Of this group, 12.5 per cent had monocular con-

vergent squint, 5 per cent alternating convergent squint and 7.5 per cent monocular divergent squint.

The results secured in this control group and in the treated group are classified with reference to the correction of the angle of anomaly and the number of patients securing binocular vision and fusion. If the angle was decreased over a period of one month, the patient was recorded as improved; if absent over a similar period, he was recorded as recovered. Of the 40 patients (table 2), 7.5 per cent were worse; 47.5 per cent were unimproved; 25 per cent were improved, and 12.5 per cent showed no squint with glasses but squint without glasses, whereas 7.5 per cent showed no squint with or without glasses. Of these 40 control patients, 60 per cent secured some degree of binocular vision, and of these 8 patients, or 20 per cent, secured fusion and stereoscopic vision. Of the unimproved patients, 11 of the 19 were in the 8 to 12 year group, while only 3 were in

TABLE 2.—*Results in Series I (Controls)*

	Objective Results					Subjective Results				
				Squint Absent			Binocular Vision			
Ages	Squint Worse	Squint Unimproved	Squint Improved	With Glasses	Without Glasses	Pseudobinocularism	No. 1	No. 2	No. 3	Fusion
2- 4.....	1	3	3	2	2	1	3	4	1	1
5- 7.....	..	5	5	2	1	5	2	8	5	5
8-12.....	2	11	2	1	0	0	1	0	2	2
13-21.....	0	0	0	0	0	0	0	0	0	0
Totals.....	3	19	10	5	3	6	6	12	8	8

the 2 to 4 year group. Equally interesting is the fact that 8 of the 10 improved patients were 7 years of age or younger, and of those patients in whom the angle of anomaly was absent, only 1 of the 8 was older than 7 years. All of the patients with squint who were improved and relieved showed 15 degrees or less of squint with glasses, and had cases of monocular convergence. None of the patients with fusion possess any fusion amplitude to date. The average time of observation of this control group was five months.

In the patients who were not operated on and were treated by orthoptic training, the refractive errors were corrected and worn six months before active treatment was begun. The youngest patient treated in the orthoptic clinic was 2½ years old. Stereoscopic fusion developed in a patient 39 years old after five postoperative orthoptic treatments, and in 5 private patients of Dr. Gifford over 25 years of age a similar result was obtained. The shortest duration of treatment was one month; the longest, one year; the average, five months. The shortest period of orthoptic treatment was fifteen minutes; the longest, two and one-half hours, the average being one hour.

In order that this report may be checked by similar clinical work, it is important that a similar routine be attempted. At the first visit the routine examination and the diagnosis are made, and atropine (1 drop of 1 per cent solution three times a day for three days) is ordered to be instilled in the fixing eye. On the second visit, the patient's angle of anomaly and his ability to fix binocularly and to see binocularly are noted and recorded. Fusion ability is determined by the pointing test, the A and E series of Wells being used in the stereoscope. The methods for stimulating binocular vision have been given elsewhere.

By using larger pictures before the suppressed eye and prisms before the fixing eye, the A series can usually be fused or superimposed in one-half hour. Sometimes the addition of from 40 to 60 prism diopters is necessary to produce fusion. Gradually the strength of the prisms is decreased until the patient is able to fuse without them. The total time consumed is one hour. If any suppression persists in this training, 1 drop of 1 per cent solution of atropine is used in the fixing eye.

On the third visit, fusion training of flat pictures (charts A to E of the Wells series) is repeated, prisms base out being used for convergent squint. The flat fusion ability is trained and stimulated at each visit before stereoscopic training is undertaken, even when good fusion is present. Check charts, especially *C*3, are always used, the patient being asked to read rapidly. If poor fusion ability is present, suppression of one eye is shown. The fixing eye is immediately found with *C*3 because only the numbers before the fixing eye are read.

Stereoscopic fusion is next developed, prisms base out being used for convergent squint before one or both eyes. Two methods of procedure are used, the interrogative and the instructive. The patient is asked to describe what he sees, and then is told what he should see. In this way he learns to fuse quickly and, of course, learns all of the Wells charts. The Sattler charts are used next, being alternated once as a check measure.

At the third period of training most patients fuse stereoscopically. This stereoscopic ability is developed for the next four periods of treatment by checking flat fusion and then simple stereoscopic fusion. Finally, after the seventh week, the most difficult Sattler charts can be fused accurately.

The next period is the training of fusion amplitude, which usually extends over six weeks. This is developed by moving the Sattler charts toward the lenses and away from them, and varying the distance between the charts at frequent intervals.

Fusion amplitude is further improved by using prisms before one or both eyes. The remaining phorias are then treated, especially during the next two months. Duction power is finally developed to overcome any remaining muscular deficiencies, Sattler's charts with prisms being

used. When the squint is corrected and does not reappear when the glasses are removed, the duction power, the phoria and vision are recorded. The patient is asked to return in one month, or earlier if the squint reappears.

If the squint reappears for short intervals or suppression of one eye is discovered with the check charts, another month of fusion amplitude and duction training is given.

This routine, if used, should secure results similar to those obtained at the orthoptic clinic of the Northwestern University Medical School.

Of the 38 patients not operated on (treated; table 3), 89.5 per cent had convergent squint, 7.5 per cent of these being the alternating type. The remaining 10.5 per cent had divergent squint, 5.2 per cent of these

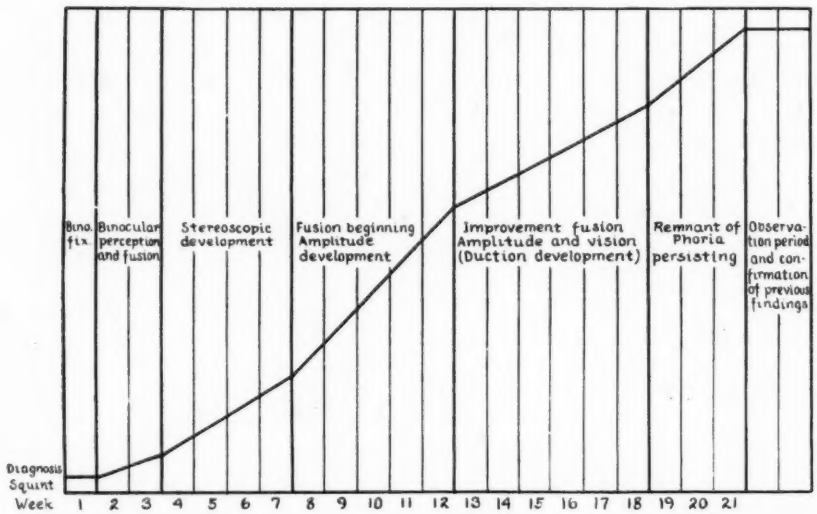
Bino. fix.
Binocular perception and fusion
Stereoscopic development
Fusion beginning Amplitude development
Improvement fusion Amplitude and vision (Duction development)
Remnant of Phoria persisting
Observation period and confirmation of previous findings
Diagnosis Squint
Week 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21

Chart showing progress during reorganization period of average convergent squint.

being of the alternating type. Monocular squint ranged from 10 to 35 degrees, averaging 30 degrees. The 5 to 7 year age group contained 84.61 per cent of monocular convergent squint of which 15.39 per cent were alternating. It will be seen that of these treated patients 36.9 per cent were 7 years or less and that 63.1 per cent were above 7 years of age.

Of these subjects (table 4), only 1 became worse; 34.2 per cent were unimproved; 13.8 per cent showed improvement in the angle of anomaly over 5 degrees, and in 50 per cent the squint was absent with glasses. Of these 19 patients, 3 had over 5 degrees of squint without glasses and poor fusion amplitude but excellent stereoscopic ability. The others showed no squint with or without glasses. So in 42.1 per cent complete relief of squint was secured even when glasses were removed.

The total time of treatment varied between two months and ten months, the average being four and six-tenths months in the cases in which treatment was given.

Among the cases in which complete relief was obtained, those of monocular convergence of low degree are in preponderance, although 1 patient with alternating convergence showed no squint after nine months of orthoptic treatment. Cases of alternating divergence, however, can be included as types of squint which readily improve.

In comparing the tables, a large percentage of cases are seen showing stereoscopic fusion at some time during the treatment. For

TABLE 3.—*Series II: Diagnosis and Age Groups in Treated Patients Not Operated On (Thirty-Eight Cases)*

Ages	Monocular Convergent Squint	Alternating Convergent Squint	Monocular Divergent Squint	Alternating Divergent Squint
2- 4	1	0	0	0
5- 7	11	2	0	0
8-12	16	1	0	1
13-21	3	0	2	1
Totals	31	3	2	2

TABLE 4.—*Results in Series II, After Treatment*

	Objective Results						Subjective Results			
				Squint Absent			Binocular Vision			
Ages	Squint Worse	Squint Unimproved	Squint Improved	With Glasses	Without Glasses	Pseudobinocularism	No. 1	No. 2	No. 3	Fusion
2- 4	0	0	0	1	1	0	..	0	0	0
5- 7	0	4	2	7	5	0	..	3	8	8
8-12	1	5	3	9	8	1	..	1	10	10
13-21	0	3	1	2	2	1	..	3	1	1
Totals	1	12	6	19	16	2	..	7	19	19

example, binocular macular perception was secured in all cases with or without the aid of prisms, while persistent stereoscopic fusion was produced in 78.8 per cent, or 32 cases, of series II. In all cases, however, some weak stereoscopic ability could be obtained at times, so that in no instance was a total lack of fusion sense present. All patients in whom the squint was absent after treatment had excellent stereoscopic ability. A similar ability was present in the patients operated on (series III).

Visual improvement is sometimes said to be essential if the squint is to be relieved and if fusion is to be developed. Only 25 per cent of the cured patients had a definite improvement of visual acuity in the amblyopic eye. Perhaps this was because occlusion was not used during the treatment.

The series of 10 patients operated on, with some squint remaining after operation, are included in this paper for completeness. No conclusions can be drawn from such a small number. Of these cases (table 5), 100 per cent were of convergent squint, 30 per cent of the alternating type. The results secured by orthoptic treatment (table 6) were 10 per cent complete failures, 40 per cent partial recovery and 50 per cent complete recovery.

If this series in which operation was performed is included in one's discussion (table 7) the total series would include 48 cases with 29.25

TABLE 5.—*Series III: Diagnosis and Age Groups in Patients Operated On and Treated (Ten Cases)*

Ages	Monocular Convergent Squint	Alternating Convergent Squint	Monocular Divergent Squint	Alternating Divergent Squint
2- 4	0	0	0	0
5- 7	3	1	0	0
8-12	2	1	0	0
13-21	1	1	1	0
Totals	6	3	1	0

TABLE 6.—*Results in Series III, After Treatment*

	Objective Results					Subjective Results				
				Squint Absent			Binocular Vision			
Ages	Squint Worse	Squint Unimproved	Squint Improved	With Glasses	Without Glasses	Pseudobinocularism	No. 1	No. 2	No. 3	Fusion
2- 4	0	0	0	1	1	0	..	..	1	1
5- 7	0	0	2	2	2	0	..	..	2	2
8-12	0	1	2	0	0	1	..	1	..	0
13-21	0	0	0	2	2	0	..	..	2	2
Totals	0	1	4	5	5	1	..	1	5	5

per cent failures, 27 per cent partial recovery and 43.75 per cent complete recovery. It will be noted that in total recovery the squint is absent without glasses. The comparison of these results (table 7) with those of others who include cases in which operation was performed discloses that total recoveries range from the 26 per cent reported by Onfray to the 80 per cent obtained by Javal, the average being 44.33+ per cent.

The results of ophthalmologists who have used orthoptic treatment alone (table 7) range from the 68 per cent of Hesse [16] to the 13.25 per cent of Aubineau,[17] the average being 46.75 per cent. The total recoveries I secured by orthoptic treatment alone were 42.1 per cent.

In comparing the control series and the series in which operation was not performed (table 8), one is impressed by the results secured

by atropinization (with definite indications) following the correction of the refractive error. In 7.5 per cent a satisfactory cosmetic result of less than 5 degrees of squint without glasses was secured; the squint has not recurred up to this time. Poor stereoscopic fusion and poor fusion amplitude were present. I realize the imperfections existing in the methods employed. If one watches the movements of the eyes through the lenses of the stereoscope and has the same check system as has been used in this work, accurate results may be obtained after several observations.

I am interested in any orthoptic technic which will yield the most recoveries in squint. However, the factors which tend to produce failures should be known. I shall omit such obvious ones as lack of

TABLE 7.—*Percentage Records in Orthoptic Training*

Author	Number of Cases*	Squint Present After Operation	Squint Absent	Binocular Vision		Stereoscopic Vision	Failure	Recovery	
				No. 1	No. 2			Partial	Complete
Griffith.....	S. 15 (alt.)	4	3	3	5	3	17%	50%	33%
Coulter.....	S. 5 (alt.)	2	3	5	5	5	0	40%	60%
Goar.....	S. 5 (alt.)	No report	2	No report	No report	2	60%	....	40%
Javal.....	S. 10 (alt.)	0	8	..	2	8	0	20%	80%
Alabaster.....	22 5 (op.)	..	6	..	..	..	33%	40%	27%
Onfray.....	100	..	..	..	75	..	....	....	26%
Guibor.....	58	10	0	..	..	42	29.25%	27%	43.75%
	38	..	16	..	..	32	34.2%	23.7%	42.1%
Peter.....	50% if below 7 years								
Onfray.....	Divergent hypermetropes 33% if below 15 years of age Divergent emmetropes 50% if seen in infancy								
Aubineau.....	37.5% in convergent squint; 13.25% in divergent squint								
Hesse.....	S. 68%								
Sattler.....	65%								

* S. indicates surgical intervention plus orthoptic training.

interest and poor application on the part of the ophthalmologist. No one can secure results by criticizing, scolding or scaring patients, or by expressing doubt in the efficiency of a method before the patient. The less obvious factors, however, which produce partial or complete failures are: (*a*) inability of the patient to alternate and to fix binocularly, (*b*) pseudobinocularism, (*c*) abnormal correspondence or projection, (*d*) persisting suppression of one eye, (*e*) lack of variation in the stereoscopic charts and cards employed, (*f*) lack of a check system, such as check cards and pointing cards, (*g*) training periods of too short duration and (*h*) poor or absent fusion amplitude and duction.

It is unnecessary to discuss these causes of failures at this time, as this has been done previously with the exception of abnormal correspondence. This condition has not been studied accurately in this series up to the present time, so that nothing will be said concerning it.

TABLE 8.—*Results in Patients Treated; Series II and III* *

	Patient	Age	Age at Onset	Squint Without Glasses, Degrees	Squint With Glasses, Degrees	Vision Without Glasses	Vision Unimproved	Binocular Vision	Stereoscopic Fusion During Treatment	Visit Fusion Occurred	Final Results	Length of Treatment, months	Comment
Worse	R. L.	11	6	R. C. 20	15	20/40 20/200	...	+	0	0	Alt. C. 45	2	Disorganization period
Unimproved	A. R.	5	9 mo.	L. C. 15	10	20/20 20/50	...	+	+	0	Alt. C. 20	1	
	M. G.	7	1 mo.	L. C. 20	20	20/25 5/200	0	+	+	3d trip	L. C. 20	2	Vision unimproved
	B. K.	8	8 mo.	L. C. 30	20	20/50 20/200	0	+	+	3	L. C. 20	8	Coloboma of macula
	J. S.	13	2 mo.	L. C. 45	35	20/25 20/30	0	+	+	0	Alt. C. 35	5	
	J. L.	5	2	L. C. 15	5	20/20 20/50	0	+	+	4	L. C. 5	1	
	S. M.	13	1½	L. C. 25	15	No record	No record	+	+	4	Alt. C. 20	1	Disorganization period
	J. N.	13	2	L. C. 15	5	20/20 20/100	+	+	+	2	L. C. 10	2	
	S. O.	7	4½	L. C. 35	35	20/50+2 20/200+1	0	+	+	3	L. C. 35	1	
	G. C.	8	4	L. C. 25	10	20/15 20/30	0	+	+	0	L. C. 10	1½	
	M. F.	9	2	L. C. 35	30	20/20—2 20/65	+	+	+	5	L. C. 30	4	
	J. N.	10	2	R. C. 15	15	3/200 20/30	0	+	+	4	R. C. 15	2	
	W. D.	9	3	Alt. C. 30	25	20/25 20/25	0	+	+	No record	Alt. C. 25	1	Alternating squint
Improved squint more than 10 degrees	F. F.	5	3	Alt. C. 40	40	20/20 20/20	0	+	+	0	Alt. C. 40	14	
	A. K.	8	7½	Alt. Div. 10	10	20/15 20/15	0	+	+	1	Alt. Div. 5	1	Poor fusion amplitude
	B. R.	15	...	Alt. Div. 30	25	20/15 20/20	0	+	+	3	Alt. Div. 5	1	No fusion amplitude
	R. W.	7	2	R. C. 35	25	20/100 20/30	0	+	+	4	R. C. 15	3	
	D. L.	12	...	L. C. 30	25	20/13 20/60	0	+	0	4	R. C. 15	3	
	L. N.	8	4	L. C. 35	25	20/13 20/25	0	+	+	5	L. C. 15	3	

Squint absent with glasses	A. E.	5	1½	L. C. 10	10	20/40 20/100	0	+	+	3	No squint	3	
	P. R.	9	1½	R. C. 40	15	20/60 20/15	+	+	+	2	No squint	2	Poor fusion amplitude
	H. B.	12	2	R. Div. 25	15	20/100 20/25	+	+	+	2	No squint	9	
	J. D.	7	1½	Alt. C. 25	15	20/50 20/50	+	+	+	3	No squint	9	Poor fusion amplitude
	V. T.	15	...	L. Div. 15	15	20/15 20/20	0	+	+	2	No squint	3	
	B. S.	5	6 mo.	L. C. 15	10	No record	No record	+	+	2	No squint	2	Poor fusion amplitude
	E. U.	8	4	L. C. 10	5	20/15 20/100	0	+	+	2	No squint	5	
	J. B.	10	1	R. C. 20	10	20/100 20/20	+	+	+	No record	No squint	5	
	E. M.	8	5	R. C. 10	5	20/50 20/40	+	+	+	2	No squint	8	
	L. N.	8	16 mo.	L. C. 15	10	20/30 20/100	0	+	+	2	No squint	6	
	G. N.	6	1½	R. C. 35	20	No record	..	+	+	3	Less than 5	2	
	A. P.	5	2	L. C. 25	10	20/40 20/200	0	+	+	3	No squint	4	
	L. P.	7	1	R. C. 10	5	20/100 20/20	0	+	+	1	No squint	5	
	L. R.	12	3	R. C. 30	20	20/50 20/20	0	+	+	No record	No squint	6	
	J. T.	11	5	R. C. 30	25	20/200 20/13	+	+	+	3	No squint	10	
	A. M.	7	1	L. C. 30	20	20/15 20/20	0	+	+	2	No squint	2	Poor fusion amplitude
	J. E.	10	5	L. C. 10	5	20/20 20/200	0	+	+	4	No squint	3	Poor fusion amplitude
	M. K.	3	2	R. C. 10	10	20/65 20/65	0	+	+	3	No squint	3	
	R. L.	14	4	R. C. 20	15	20/50 20/13	0	+	+	3	No squint	4	

TABLE 8.—*Results in Patients Treated; Series II and III* *—*Continued*

	Patient	Age	Age at Onset	Squint Without Glasses, Degrees	Squint With Glasses, Degrees	Vision Without Glasses	Vision Improved	Binocular Vision	Stereoscopic Fusion During Treatment	Visit Fusion Occurred	Final Results	Length of Treatment, months	Comment
Operative plus orthoptic training	W. P.	8	3	R. C. 15	10	20/40 20/30	+	+	+	2	Squint 5	2	Improved
	A. K.	13	8	R. C. 10	10	20/20 20/13	+	+	+	2	No squint	4	Squint absent
	J. N.	12	2	L. C. 15	10	20/15 20/100	+	+	+	1	L. Con. of 10	2	Unimproved
	F. P.	7	...	L. C. 15	10	20/15 20/50	+	+	+	4	No squint	1	Squint absent
	C. M.	9	4	L. C. 15	10	20/50 20/40	No record	+	+	5	No squint with glasses, without 10	2	Improved
	A. M.	7	1	L. C. 30	20	20/15 20/20	+	+	+	2	No squint	2	Squint absent
	S. K.	6	5 mo.	Alt. C. 25	25	18/200 20/30	+	+	+	16	Alt. C. 10	6	Improved
	H. M.	7	6 mo.	Alt. C. 20	10	20/15 20/15	—	+	+	1	L superaver-gence 10	4	Unimproved
	M. W.	16	6	R. C. 15	15	20/13 20/13	—	+	+	1	No squint	1	Squint absent
	L. S.	4	7 mo.	Alt. C. 10	10	No record	—	+	+	2	No squint	1	Squint absent

* R. C. indicates right convergence; L. C., left convergence; Alt. C., alternating convergence; Alt. Div., alternating divergence; R. Div., right divergence; L. Div., left divergence.

CONCLUSIONS

A preliminary report is offered of the possibilities presented by orthoptic training of nonparalytic squint in 38 unselected cases of strabismus compared with the progress made during the same period in 40 cases in which the patients were treated by the usual methods but without orthoptic training. A subsequent report will be made of the future progress of this series and of additional cases.

In series I, fusion and depth perception were developed in 20 per cent of the cases. Improvement in the angle of squint of over 5 degrees was obtained in 25 per cent, and complete correction of the squint occurred in 7.5 per cent. In series II (the treated patients) binocular macular perception was obtained in all cases at some time during the treatment, and fusion in 99 per cent. Complete correction of the squint was obtained in 50 per cent of the cases with glasses and in 42.1 per cent without glasses. The improvement in vision was seldom marked. More improvement might have been seen in younger patients subjected to prolonged monocular occlusion of the fixing eye as a preliminary to orthoptic training. In 7.5 per cent of the cases, although fusion was obtained, the angle of squint was not improved during the treatment. It is suggested that the value of orthoptic training can be realized only by the observation of patients treated under systematic supervision by persons especially interested in such training. Special clinics offer distinct advantages in many cases. The length of each period of training is important: The treatment, which varies from fifteen minutes to two and one half hours, should be continued as long as the patient is improving. The attention of the average patient can be kept for one hour by proper variation of charts and procedures. Training at home is a distinct aid but is not essential to obtain results. Most of the patients in this series had no such training. Three patients with alternating squint had fusion, one showing a slight improvement in the angle of anomaly. Hence I do not agree with Worth and Peter that a congenital absence of fusion exists in these cases, so that they are unsuitable for training. There seemed to be no age limit in these cases beyond which it was impossible to develop fusion. Some patients between 2 and 4 years of age cooperated sufficiently for training and for accurate observation in the control series. Patients showing considerable amblyopia were capable of developing fusion in the stereoscope.

ABSTRACT OF DISCUSSION

DR. LUTHER C. PETER, Philadelphia: It is a real pleasure to discuss Dr. Guibor's preliminary report of a year's experience in orthoptic training. I congratulate him on this work and hope that others may be induced to make similar studies and be convinced as to what can be accomplished by systematic training in concomitant squint.

In this discussion one should carefully differentiate between true alternating squint and the monolateral variety. They have little in common other than the manifest deviation, and they are different entities. If visual acuity is equal in the two eyes, or after visual acuity is restored, monocular squint often becomes alternating. It then may be difficult to decide that the case was essentially monolateral. True alternating squint occurs at birth or during the early part of the first year. Refractive errors are negligible, or, if considerable, are of equal degree. In my experience, fusion is not present and cannot be developed. If fusion can be developed, the case immediately falls into the category of monocular squint. Amblyopia obviously does not occur. If the family history is carefully examined, it in itself will help in the classification.

A second premise or condition that should be clearly grasped in the beginning of such study is the ability to resolve orthoptic treatment into its component parts. Strictly speaking, orthoptic training should be limited to the development of single binocular vision with stereopsis. Practically, however, this end is obtained only by restoring vision within useful limits, and by overcoming the habit of suppression. Speaking broadly, therefore, orthoptic training is made up of three steps:

1. The restoration or preservation of vision in the squinting eye.
2. The breaking up of suppression.
3. Fusion training.

In my experience, if the first and second barriers are removed and fusion is present but weak, fusion training really becomes a simple matter and can be obtained in a comparatively short time. In many cases it automatically develops with little artificial stimulation.

Amblyopia is the great problem. To place myself on record (since I have been misquoted frequently), my belief is that there is no definite upper age limit in the correction of amblyopia. Until the seventh year, most cases can be restored to normal vision with proper methods; from 7 to 12 the percentage of recoveries is slightly lower; from 12 to 21 years the possibilities are still fair; after 21, 6/9 vision is rarely obtained by training, and this vision has probably been reached in childhood or early adolescence.

Last year I reported three groups of cases at the Oxford Congress:

Group I: Thirteen patients, with an average age of 5½ years. Average vision before training, 6/15; after training, 6/6.

Group II: Seven patients, with an average age of 11 years. Average vision before training, 6/14; after training, 6/7.

Group III: Ten patients, with an average age of 17½ years. Average vision before training, 6/14+; after training, 6/7.5. The oldest patient treated was 21 years of age and was corrected from 6/15 to 6/7.5 partly.

In my experience the average time required to restore vision is about four months. It can be corrected in most cases in three months or less, but there are many lapses in an average case when cooperation lags or sickness intervenes.

As to the relative value of occlusion and atropine, my experience teaches me that occlusion is better and incurs less risk. In Claud Worth's sixth edition of his monograph, page 111, he says: "But, of course, continuous occlusion of the fixing eye would produce the same results [as atropine] very much more quickly." This is my experience.

In children of school age, occlusion is ideal if the teacher's cooperation can be obtained, and I find that the average teacher is most willing to cooperate. There are objections to atropine which my time will not permit me to discuss.

Intermittent occlusion, in most instances, and complete occlusion in selected cases, if the proper occupation at the near point is selected, accomplishes maximum results in a minimum of time, with few disadvantages and no dangers.

Whether suppression shall be allowed to continue or whether it shall be corrected may be determined by a careful classification of each patient as to the type of squint. After eighteen years of special work and study in squint, I have come to the conclusion that true alternating squinters are without a fusion faculty, that this defect is found in other members of the same family, and that it is better to allow the habit of suppression to continue throughout life in the interests of comfortable vision.

If fusion can be elicited even in crude form, suppression should be corrected and fusion training should be instituted.

Suppression can be interrupted at the beginning of treatment by flashing on the synoptophore and by the same process on a rotating stabilizer. In the former, adjustment is made for the degree of squint by bending the tubes to the proper angle. On the latter instrument, correction is made by prisms after the rotating light has awakened monocular fixation in the amblyopic eye. On the latter instrument the target is larger and macular stimulation is rendered easier. Both these instruments not only stimulate fixation and tend to break up suppression but pave the way for fusion.

The actual orthoptic treatment should begin with the amblyoscope or synoptophore. The latter is preferable, is under perfect control of the operator, and eliminates the uncertainties of the stereoscope.

After proper office or clinic training with these instruments, the stereoscope should be added for home treatment of fifteen-minute sessions twice daily. Prisms may be needed, and in this work the average mother can be trained. I do not feel that three periods a week are sufficient to develop a well balanced fusion that will stand the rigid tests of stereopsis.

Dr. Guibor has pointed out the necessary steps in this training. He also stresses the difficulties. These difficulties can be corrected on the synoptophore. If the Hamblin, Wells, Sattler or Keystone cards and views are carefully followed, even the home use of the stereoscope can be carried out correctly.

In my squint clinic in the Graduate School of the University of Pennsylvania, the nurse in charge trains the mothers as well as the children and succeeds well in this type of case. In private practice, one of my office force does the same with equal success.

In the early part of this discussion I stated that "fusion training is easy in most cases." In awakening this faculty when it is present there are no upper age limits, either in children or in adults. In adults, however, the decision as to whether one shall allow suppression to continue or whether fusion training shall be pushed to its limit must be made only after carefully weighing the evidence in each case as to which method is apt to yield the most comfort for the patient.

Finally, in my work, most of which is in private practice, my belief is that: 1. Glasses alone, when prescribed early, will straighten the eyes to a degree at which fusion training can complete the cure in 50 per cent of cases, if the squint is not over 25 degrees. 2. In squint of more than 25 degrees, operative help will be needed to complete the cure in most instances. 3. The correction of amblyopia in children up to 7 is possible in most instances. Failures can be attributed in a few instances to acquired pathologic change of the macula, either visible or invisible. Failures in most instances can be attributed to incorrect methods of training or to a lack of cooperation. Each year after 7 up to 20 offers increasing difficulty in effecting

cures. 4. Fusion training will succeed in most cases at any age in monocular squint if operative measures are properly carried out in those who are in need of them, and in the nonsurgical cases, if orthoptic training in its broadest sense has been systematically and painstakingly applied.

DR. DAVID W. WELLS, Boston: The value of such studies on orthoptic training cannot be overestimated. Only thus can there be accumulated reliable data of the best methods of treatment.

Not many years ago it was common for the family doctor to advise the parents that the child would outgrow the "squint." Just what proportion recovered spontaneously would be interesting to know. It is with such a series that the present report should be compared.

The series at the Children's Memorial Hospital, used as a "control," consists not of untreated patients but of the partially treated. The treatment given was the recognized early handling of every case: measurement of refractive error under atropine; constant use of correcting glasses; the fuller correction of the fixing eye (my own practice is to use bifocals), and atropine in the fixing eye, which, the author quotes Worth as saying, is "an exceedingly efficient curative measure." The weekly tests continued five months to determine progress are also treatments. In this series of 40 cases, "25 per cent improved, 12 per cent no squint with glasses on, 7 per cent no squint without glasses." This is certainly no mean result. In the personally trained series, "50 per cent showed no squint with glasses, 42 per cent no squint without glasses." The value of personal training is so definitely proved that it should be called settled. I have not made a detailed analysis of my own cases for many years, but I doubt that it would show this percentage of cures. I can find little to criticize in the author's technic. I believe the lesson at the doctor's office should be followed by home training and that fusion amplitude is best cultivated by the decentration of spheres in the phoro-optometer, with the fused image as an anchor.

The question as to how much can be done for amblyopia is of great interest. Twenty-five per cent of the cured cases show definite improvement. I hope the author will tell more exactly what "definite" means. I have never been able to accomplish as much in this line as I had hoped—a few phenomenal results, but with the majority an improvement of only two or three lines on the chart. I have been waiting to see the reports of those who have used the cheiroscope, which is based on the mental training of the poorer eye by the image seen with the fixing one. There is little hope of materially improving the vision of amblyopia, except that part due to exanopsia.

The author tests fixation by watching the child's eyes through the stereoscope lenses. By cutting out the top of the hood, one may view the eyes directly.

The author is to be congratulated on an important piece of work, well done, and well reported.

DR. SANFORD R. GIFFORD, Chicago: I have one or two corrections which should be made in Dr. Guibor's report. One is that the series of operative cases did not include all of our operative cases but only 10 cases in which some squint remained. I should like also to mention the fact that we have a grant from the Academy which is helping us in the work which we expect to continue, and which should include some very careful studies of depth perception. Aside from my interest in Dr. Guibor's problem and his report on it, I am glad to discuss it for several reasons. One should realize that this is the first report yet to be made on a series of cases following orthoptic training in which enough details were given to show exactly what results were obtained in individual cases and in the group

as a whole. I believe Dr. Guibor's report shows very fairly what can be done in a similar group under similar training. I think one must admit that most patients with squint should be given the benefit of such training, either before operation is undertaken or afterward, if operation is required. I speak with feeling on this point, as I approached this subject rather skeptically, with the idea that most patients with squint were so trained in suppression of one image that not even a beginning could be made with them. One tried the amblyoscope, and if fusion was impossible at the first attempt, very often no further attempt was made. From the present report it is evident that nearly every person with squint is capable of some degree of binocular vision, so that a beginning can be made in fusion training.

I do not believe that orthoptic training will replace operation in the treatment of squint. It will be seen from Dr. Guibor's tables that the best results were chiefly in squint of less than 20 degrees. In extreme cases a great deal of time will be saved by an early resort to operation, after the results of refraction and a short course of training have been ascertained. It would seem that if, after three to six months of training, no further progress is being made, operation should be advised. This should usually be followed by further training until binocular vision becomes established.

With clinical material, most of the training will need to be done in the clinic, and some such system as we have employed at Northwestern University would seem to be the only practical way of meeting the situation.

What all should be interested in is the practical use of orthoptic training in private practice. Here one can usually count on more intelligence and cooperation from the patients or their mothers, and a routine can be outlined which will greatly lessen the number of office visits required. It does not pay to count too much on maternal aid, however, and in the case of very young children it often occurs that training may be begun only by an expert both in the technic of training and in child psychology. The busy practitioner will often be only to glad to refer such cases to an expert trained for this work, checking progress personally at proper intervals. Once a start has been made, further work by the patient with or without supervision by the mother is usually possible. Office equipment need not be elaborate for this and need consist only of an amblyoscope, a stereoscope, the Wells and Sattler charts, and a set of prisms for a small trial frame, which can be worn when the stereoscope is used. I have found the cheiroscope of Maddox and the synotophore of value in starting some patients who could not fuse with the other instruments. The Keystone drawing charts with which the patient draws a fused image are not of value until some fusion is present but then offer a great stimulus, especially in the case of children, to continue training and seem to be of definite benefit.

Cases of intermittent squint are especially suitable for orthoptic training in the office and at home. I have had 2 cases in children under 6 during the past year in which such training has resulted in habitual fusion, correct depth perception and entire disappearance of the squint, and a number more are showing improvement. Such results of course may be seen after refraction alone. One is often told by mothers that the child squints at times, but examination at the office discloses no manifest squint. It gives both physician and parent a good deal of comfort if tests for fusion are done in such cases, as these may reveal normal fusion or a monocular suppression that may be treated successfully before a real squint develops.

Of 12 private operative cases in which subsequent orthoptic training was given, fusion was obtained in all, and in all but two depth perception was good. The ages ranged from 6 to 26, and in 2 patients over 20 who had squinted since early childhood good fusion was obtained.

One woman of 23, with a macular coloboma reducing vision to 3/200 in the eye that had been operated on, much to my surprise could fuse the larger Sattler charts with correct depth perception. In several of these cases a sqint of from 5 to 10 degrees remained after operation, and this cleared up entirely in all cases but 1. This was a case of anomalous correspondence in which the cosmetic result is excellent, but on covering of the fixing eye the eye that had been operated on turns out to fix. This patient is under training at present.

One case will illustrate the effect of fusion training on a postoperative remnant of squint. Emily P., aged 12 years, had had a right convergence of 25 degrees since the age of 3. Following operation (tucking of the right externus and recession of the right internus) the eyes were straight, but three weeks later a convergence of 10 degrees was present. Fusion was impossible with the stereoscope or amblyoscope. With the Maddox cheiroscope, however, solid objects could be fused. After this had been used at home for a week, the stereoscope could be used with seven prisms bases out. She was given such prisms in slip-overs to use with a stereoscope and the Wells charts. Two weeks later fusion was possible without prisms; but convergence of 10 degrees was still present. After four weeks more fusion was possible while overcoming 14 degrees of prism base in, and this was given for the exercises. Convergence of 5 degrees was still present, but immediately after exercises it was absent. After two more weeks the eyes were straight for distance and near on cover test. The base-in prisms were increased to eight base in for each eye. Two weeks later the eyes were straight and most of the Wells charts were fused with proper depth perception. Three months later the condition was the same, except that all the charts could be fused, and objects in the battle-ship chart for depth perception were judged with an error of only 10 per cent.

Such cases demand a good deal of time and not a little thought. It is obvious that without such time and thought good results cannot be expected. One meets with disappointments in some cases which appear favorable. I have a girl of 5 at present with only a moderate squint in whose case persistent effort has failed to obtain any fusion. On the whole, however, the results have been surprisingly good and I am sure that they have been well worth the effort expended.

DR. CONRAD BERENS, New York: Ophthalmologists are indebted to Dr. Guibor for his scientific study of orthoptic training. Because of the great importance of this work, the section may be interested in the work done in New York state during the past year. Over a year ago, the New York State Commission for the Blind organized clinics for orthoptic training throughout the state. These were most successful because the training was done by a woman who knew how to organize the work of the children and who was familiar with the principles of orthoptic procedures. The National Society for the Prevention of Blindness is especially interested and may be willing to train laymen to do this type of work if ophthalmologists believe it advisable. As is well known, it is impossible for the busy ophthalmologist to do this work himself, and he should have a trained assistant in his office to help him. I should like to know from the members of the section whether they believe that it is advisable to train laymen or trained nurses to do this work. A young woman trained in psychology did this work in my office and is now devoting her entire time, in New York City, to orthoptic training and perimetry as a profession. What are some of the factors that prevent successful results in orthoptic training? Lack of proper organization is the greatest handicap. Trained personnel and proper equipment are necessary, and the limitations of the various methods of training must be known. Too many expensive instruments have

been constructed and new trade names given to simple apparatus. Cheap useful stereoscopes are available and valuable work can be accomplished with a piece of red glass and a candle. Dr. Wells has shown the importance not only of training under proper supervision but of training at home. If clinic patients are to be trained at home, a stereoscope must be available that they can afford. Most clinic patients cannot afford even two or three dollars. There is a medical problem in many patients with strabismus which should not be overlooked. Size differences and amblyopia are important factors in preventing fusion. Size lenses and training of the amblyopic eye are of value. Probably the most important point is that many patients with squint do not improve with all the usual methods of nonoperative treatment. It is important to operate on these patients and place their eyes in position where they may be trained, as soon as it is evident that orthoptic measures will fail.

Dr. Thomas D. Allen, Chicago: I have carefully followed 1 case reported by Dr. Guibor, an alternating squint, in which combined advancement and tenotomy had been done at the age of 15 years. Good depth perception was obtained, but little amplitude of convergence or divergence. About twelve years ago a similar operation was done on a boy of 12 or 13 years of age. He had also what was diagnosed as alternate squint. Vision of both eyes was normal, with little or no correction. After operation it was found that he had depth perception with practically no training. I am obtaining fairly satisfactory results with the new Keystone apparatus.

Dr. George P. Guibor, Chicago: With such a paper as this, in the time allowed, many details had to be omitted, so these details which Dr. Wells and Dr. Peter discussed are in the original paper, including a discussion of the literature.

Regarding home training, these were all clinic patients, they were receiving relief, and so they had no stereoscopes. I felt that I could not afford to lend them stereoscopes, especially in an orthoptic clinic where there were over 100 patients; so most of this work was done in the clinic.

I wish to correct one impression, and that is in regard to the number of treatments. They were given one treatment a week not three.

As regards the amblyopia being corrected by the covering of the fixing eye, we started with the impression that covering the eye would produce better results, and so many patients were lost that there was danger of closing the clinic. So I do not cover the fixing eye until after the patient has some ability to fix with the squinting eye. I believe, from our short experience, that the indication for covering the fixing eye is improvement of vision. Perhaps the amblyopia might be improved more if it was possible to cover the fixing eye from the beginning. I do not think that improvement of vision should be recorded unless over two lines on the Snellen chart are shown.

In all of these cases in which the squint was relieved, I had the patients return to the clinic and had some one check them over. Some one besides myself could verify the fact that these patients were relieved of their squint. And that is not all. I believe that in most reports given it is stated that the squint is corrected with glasses. In these cases the squint was corrected without glasses. A few patients lost their glasses, 2 for two months, and the squint did not reappear. I have been observing them for six months.

CLINICAL DETERMINATION OF THE LIGHT THRESHOLD

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In discussing the function of visual acuity one is inclined to speak at times too casually of perception of light, of color vision and of perception and identification of form. One may fail, however, except when under the necessity of considering details of retinal physiology, to differentiate retinal function into that which is purely ocular and that which is neurocentral, even of a subcortical nature. Thus, one is vague and indecisive in separating retinal sensitivity and response into certain stages: the first, that of primitive perception; the second, that wherein there is an integration of recepts of a more highly discriminative pattern in which qualitative as well as quantitative details of the constituent sensations carry into consciousness factors such as attention and awareness, the third, a "learning by experience" stage.

The first stage can be called a dyscritic stage; it is a purely primitive one wherein no discrimination or differentiation is present. The retinal recepts concerned give rise to changes in the stream of consciousness or, as the psychologist says, of psychoplasm, of such a kind that the cenesthesis (undifferentiated complex of organic sensations which make up that spoken of as vital sense) is raised in potential, giving rise to awareness. This phototropism is the response from that microscopic pigmented spot in certain minute unicellular forms of life, e.g., Euglena virides (a flagellate), which enables them to advance or to recede from areas of greater or lesser illumination. With such an organism perception of light is a trial and error effort in the search for food and comfort. This most simple of eyes is capable only of that function which is called perception of light. The verted eye of certain forms of life such as the worm, Stylaria lacustris,[1] is another but more complex example.

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1. Wolff, Eugene: The Anatomy of the Eye and the Orbit, Philadelphia, P. Blakiston's Son & Co., 1933, p. 255.

It is wholly separate and distinct from the second or epicritic stage. Parsons [2] offers a definitive description of the latter form of perception: "The constellation of recepts [the stimulus and its ocular reactions] gives rise to a perceptual pattern in which cognitive elements are so differentiated that the diverse sensations are discriminated, awareness is focused upon those features of the pattern which are of significance and awareness becomes first attention and later experience." Thus, in terms of retinal sensation, are expressed the usual and highest form of usable visual effort and results.

In a new and exhaustive work on the measurement of visual acuity, the tasks involved [3] are discussed as threefold: determination of the minimum visible, the minimum separable and the minimum legible. This writer has somehow failed to emphasize that these tasks involve two separate forms of sensation, the first of the three being dyscritic (or protopathic), and the last two, epicritic. It is interesting that Guillery [4] would like to abolish wholly the term visual acuity because in practice it is a measurement of the minimum separable. His claim or desire is based on the fact that the theory of visual acuity is itself based on two misconceptions, the first concerning the use of geometric optics, and the second arising from inadequate experimental data, for resolution is possible at much higher angles than one might expect from the histologic and optical elements considered necessary. The visual task of judging the absence or presence of a white dot on a black background is one of pure light perception and is no different from that of seeing and in being aware of a star of minimum unaided magnitude with its infinitesimal and minute visual angle which must subtend the retinal element with its minimal amount of luminosity. The comparative surround of the two is similar. Regardless of the minuteness of the visual angle of this beam of light from dot or star, one is aware of it as long as its luminosity is adequate for arousing or stimulating the threshold of the cone or the rod on which it falls. It is seen better, or sooner, perhaps, if the element of motion, as an additional physiologic factor, is added,

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2. Parsons, J. H.: An Introduction to the Theory of Perception, New York, The Macmillan Company, 1927, chap. 4.

3. Lythgoe, R. J.: (*a*) Illumination and Visual Capacities, Medical Research Council, Special Report Series no. 104, London, His Majesty's Stationery Office, 1932; (*b*) The Measurement of Visual Acuity, Medical Research Council, Special Report Series no. 173, London, His Majesty's Stationery Office, 1932.

4. Guillery: Messende Untersuchungen über den Lichtsinn bei Dunkel- und Helladaptation, Arch. f. d. ges. Physiol. **70**:450, 1898.

for then its image sweeps back and forth across contiguous cones or rods, and further, as that portion of the retina is approached which is extramacular and in which rods exist in greater numbers, such a minimum light stimulus may be identified even more readily.

Mention of certain factors connected with the epicritic function is relevant because of comparison. If the task is to differentiate between one dot and two dots lying close together, it is a test for the minimum separable, and, lastly, if the white area has a more complicated pattern, accurate identification of which is necessary, this task falls under the head of the minimum legible. The minimum separable is a problem of optical resolving power, hence, is now in part epicritic, and depends not only on the angle between the two points but also on the intensity of their illumination or luminosity. The minimum resolving power is assumed to be (through experimental work confirmed and reconfirmed [5]) an angle of about 60 seconds. The minimum legible is still more complicated and depends not only on the resolving power of the retina but also on a physiologic function spoken of as the alining power of the retina. This is best illustrated in one's ability to see a black thread on a white background, or the reverse, and is possible with an angle as small as 2 seconds. It depends on the threshold of the light sense of the adjoining retinal cones and the type of image that is cast on the mosaic of rods and cones. One can see that the slight constant movements of the eye which are normally present change constantly and greatly the position of the image pattern on this mosaic. There are many other factors which enter into this second stage of retinal perception, but their mention is not necessary to the discussion either for emphasis or for explanation. Brief mention will be made, however, of illumination alone. Lythgoe [3] has pointed out something of great importance in regard to illumination and visual acuity. Beyond a physical and proportional increase in the brightness of the retinal image, illumination can produce only an effect on the physical side—and it certainly has never been shown that illumination, per se, has a psychologic action. It may be expected, however, to produce profound effects on the physiologic processes. This is the only reason why it has such important theoretical as well as practical bearings on visual acuity.

Considering that which I have just stated, a reason for the further study of the light threshold would seem to be important. There is no

5. Frölich, F. W., and Vogelsang, K.: Ueber eine physiologische Methode die Ausdehnung der Fovea centralis zu bestimmen, Arch. f. d. ges. Physiol. **207**: 110, 1925.

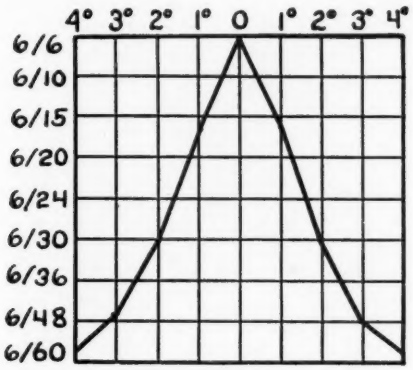

Fig. 1.—Graph showing the fall in central visual acuity as the region of the fovea centralis is passed; regional sensitivity for acuity of vision. (Redrawn after Evans.)

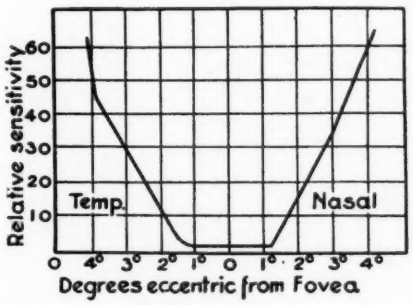

Fig. 2.—Graph showing regional sensitivity of the retina in the foveal region for mixed bluish-white light. (After Breuer and Pertz.)

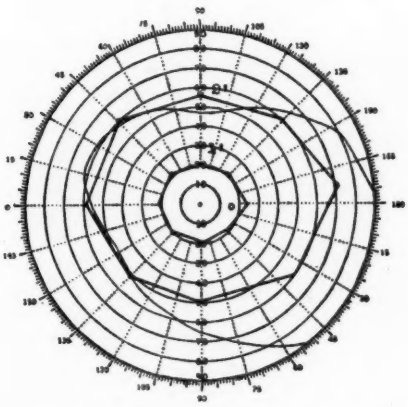

Fig. 3.—Isopters for the 1 and 2 minute test objects plotted together. (After Evans.)

doubt that the retinal anatomic distribution of the elements which subserve such a sensation is no different from that of the elements which continue toward the epicritic sense. The former elements are subject to the same defects as are those for visual acuity. Their distribution in the retina is, however, slightly different.

This microscopic, anatomic distribution of elements which have to do with central, paracentral and peripheral visual acuity (the epicritic sense) as well as bare light perception (the dyscritic sense) is rather well known, so well that it is unnecessary to include it herein. For the purpose of this paper, however, two graphs and a sketch illustrate this anatomy (figs. 1, 2 and 3). The first graph, modified after Evans,[6] was redrawn to match in scale the second, this being after Breuer and Pertz.[7] Figure 1 shows the rapid peripheral fall in visual acuity and is wholly dissimilar to figure 2, which in turn shows the rapid rise in minimum light sensitivity as the periphery is approached. Both graphs are extrafoveal studies. In the first, a standard Landolt broken-ring scale was used; in the second, a mixed, bluish-white light. The sketch (fig. 3), also after Evans, shows the isopters for the average 1 and 2 minute test objects plotted together. The three together illustrate most vividly the minuteness of the reading area of the retina. While the fovea is the most sensitive portion of the retina in the light-adapted eye, it is the least sensitive in the dark-adapted one. The paracentral region is fully a thousand times more sensitive,[8] under these circumstances, than is the fovea itself. Breuer and Pertz [7] found that sensitivity to white light was minimal over a central area of 2 degrees, but then it rose rapidly to a maximum from 10 to 20 degrees from the fovea, and that in all probability beyond this it in turn fell to some extent. The more recent papers of Henschen [9] on light and color perception cells in

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6. Evans, J. N.: A Scotoma Associated with Strabismus, Am. J. Ophth. **12**:194 (March) 1929.

7. Breuer and Pertz: Ueber die absolute Empfindlichkeit der verschiedenen Netzhautteile im Dunkeladaptierten Auge, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **5**:327, 1897.

8. (*a*) Duke-Elder, W. S.: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, p. 897. (*b*) Simon, R.: Ueber Fixation im Dämmerungssehen, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **36**:186, 1904.

9. Henschen, S. E.: Light and Color Perception Cells in the Geniculate Ganglion: II, Hygiea **92**:585, 1930; Clinical and Anatomical Contributions on the Pathology of the Brain: 8, Stockholm, published by the author, 1930; Centralbl. f. d. ges. Ophth. **23**:243, 1930; Light-Sense Cells and Color-Sense Cells in the Brain, Hygiea **91**:705, 1929.

the geniculate ganglion certainly seem to indicate the presence of highly specialized cells of the central nervous system with individual and separate functions, as well as new and hitherto unknown cerebral connections. It is, therefore, not at all improbable that the line between a dyscritic and an epicritic sense is even more sharply drawn than has been thought. Clinically, I feel that such differentiation is present.

It is interesting and relevant to illustrate the fields of vision in a case of quiet glaucoma following trephining, the field having been taken while the eye was dark-adapted. The test object used with the flicker method for the patient's peripheral field was an illumination equal in degree to that of his minimum light sense as found parafoveally. The other field was taken with a 0.5 degree test object under the common stand-

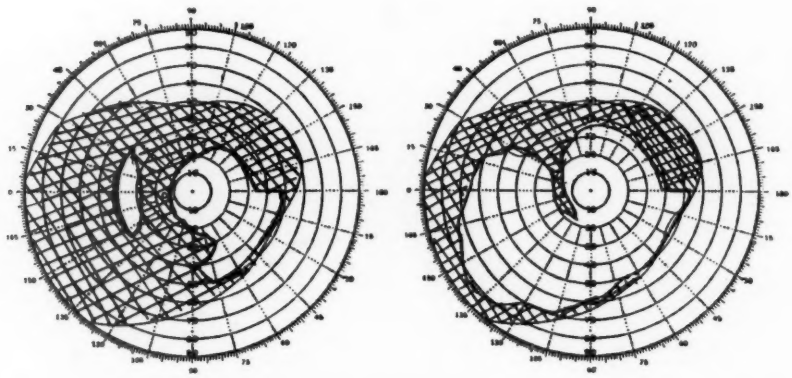

Fig. 4.—Visual fields in a case of quiet glaucoma: On the left, the field obtained with a 0.5 degree test object under standard conditions; on the right, the field obtained with the flicker method, the illumination being the patient's minimum light threshold as found with the adaptometer.

ards. The difference between the two is most interesting and significant in relation to the subject matter of this paper. It reproduces rather well, it seems, a fair picture of this patient's true minimum light sense retinal mosaic (fig. 4).

The consideration of such a twofold function, each part a definite entity, seems necessary for the explanation of many of the findings. It is certain that the light sense operates alone in certain forms of life, and it is as certain that its integrity is essential for the epicritic form of perception. The first function may be present alone, but the second, if present, proves that the first has not been lost. The various vagaries which appear between the threshold of light sense and central visual acuity are also rather significant. The central scotoma (in acuity of

vision) found in amblyopia ex anopsia is always far greater in degree than the increase in the light threshold. A patient with chronic glaucoma with 6/7.5 vision may have an increase in light minimum to 0.045. A person with secondary atrophy of the optic nerve can have a light sense of 0.90. A patient with retinitis pigmentosa with 6/9 vision will have a light minimum as high as 0.010. A patient with 6/4 vision in each eye will show a normal light minimum in one eye and one of 0.50 in the other, all other things being wholly normal. The light sense of a person with the primary tabetic type of atrophy seems more impaired in proportion to the visual acuity than the light sense of a person with postneuritic atrophy of the optic nerve. A decrease in the size of the peripheral field for any organic reason usually means an increase in the paracentral light threshold, but this is not an essential accompaniment. Central scotomas may be seen without the light sense being wholly lost, in fact, with good light sense still present. Many other clinical instances will be considered later.

ESSENTIALS FOR A SATISFACTORY PROCEDURE

Granted that for the moment every one agrees to the practicality of routine testing of a dyscritic sense and that it has true clinical value, there are a few important factors relevant to the determination of a light minimum by any method whatsoever. Most of them will be discussed in the following connected groups.

The first group includes the size of the retinal image, its position, its chromatic stimulus, the intensity of light used, the area of the pupil and a necessary correction for differences in this.

The second group should include the following: the necessity for preexposure or preliminary light adaptation and subsequent dark adaptation, dark adaptation at the fovea, the theoretical bases of or opinions relative to dark adaptation, visual acuity during dark adaptation and a standardization of adaptation.

In the third group, logically, methods of testing would be discussed as to ease of operation, standardization of the results in an easily applied scale and the value of the test from a clinical standpoint. It also should include standardization as to the length of time of exposure for the threshold illumination, and certain psychologic factors such as warnings, etc., which might be relevant, should be controlled as well. In this last group no mention will be made of the various other methods which have been used. Such a discussion would add nothing to the subject

matter. Derby [10] and his group of co-workers have already reviewed this subject sufficiently. A further brief mention will be made later.

In considering the first group, I shall first discuss the size of the retinal image. Later it will be seen that the region 40 degrees to the nasal side of the retina is the most sensitive for determination of light threshold. For obvious reasons, however, fixation in the patient must be utilized. Cowan computed for me the position of the retinal image and its maximum angular size, utilizing, naturally, the Gullstrand schematic eye, without the factor of accommodation, which herein plays no rôle. In the apparatus described in this article, considering the distance the aperture lies from the cornea, the retinal image falls well within a 19 mm. area parafoveally on the retina, as outlined on Cowan's chart,[11] and presents an area of something less than 36 degrees to each side of the point of central fixation. This should be adequate for a standardized region from which the dyscritic sense can be judged. While a round image area might be more ideal, the four angles of the square actually include a greater surface on the retina with a smaller cross-surface pupillary aperture.

Hecht,[12] with others, felt that the image should be confined to the area of the fovea, that is, in the rod-free region, and that a fixation point should be used which could be recognized for both form and color. This does not seem logical. The rate of adaptation at the fovea is different from the rate parafoveally; hence relative central scotomas appear when one considers the longer period of adaptation necessary for peripheral adaptation. Rod-free conditions with pigment effects on light absorption, as well, are considered by Tscherning [13]—these and others all present definite but incalculable factors. It is quite likely that rods and

10. (*a*) Derby, G. S.; Chandler, P. A., and Sloan, L. L.: A Portable Adaptometer, Tr. Am. Ophth. Soc. **27**:110, 1929. (*b*) Waite, J. H.; Derby, G. S., and Kirk, E. B.: The Light Sense in Early Glaucoma, Tr. Ophth. Soc. U. Kingdom **45**:301, 1925. (*c*) Derby, G. S.; Waite, J. H., and Kirk, E. B.: Further Studies on the Light Sense in Early Glaucoma, Arch. Ophth. **55**:575, 1926. (*d*) Derby, G. S.; Chandler, P. A., and O'Brien, M. E.: Further Studies on Early Glaucoma, Tr. Sect. Ophth., A. M. A., 1928, p. 37. (*e*) Derby, G. S.; Chandler and Faissler: Further Studies on the Light Sense in Early Glaucoma, in the Library of Massachusetts Eye and Ear Infirmary, unpublished owing to untimely death of Dr. Derby.

11. Cowan, A.: A Suggestion for a New Perimetric Chart, Am. J. Ophth. **3**:48, 1920.

12. Hecht, S.: The Nature of Foveal Dark Adaptation, J. Gen. Physiol. **4**:113, 1921.

13. Tscherning, M.: Adaptation of the Eye to Light, Compt. rend. Soc. de biol. **86**:223, 1922.

cones both enter into dark adaptation. Certainly one must consider Parsons' statement,[14] when he said, "the law governing the relation between threshold energy and the size of area stimulated shows no specific difference between rods and cones, and depends on the concentration of neuro-epithelial elements, etc." Figure 5 illustrates the position of the retinal image on the Cowan chart. It is interesting that the patient is not aware of individual points of light, but the entire field of vision seems filled with the sensation of light. One would expect this with an image much larger than the 5 mm. square aperture object. Also, by reason of certain fixed optical principles, this aperture, at the distance it lies, cannot give an image of multiple light spots, but one

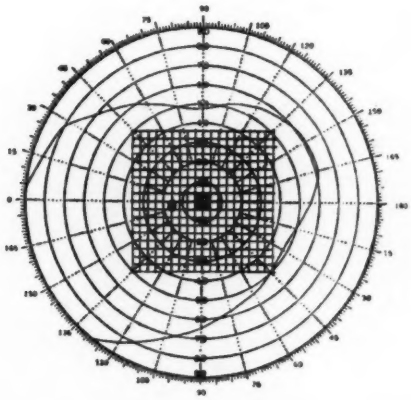

Fig. 5.—Retinal position of the aperture image.

with only a single diffuse image or stimulus. The chromatic stimulus used will be more properly presented later when the apparatus itself is being discussed. The intensity of light used was first gaged from the normal by simple trial and error. When a satisfactory minimum was found, further standardization was possible. With rheostatic control, this can be easily changed at will. It is necessary, however, to know the candle-power being delivered under such changed conditions and to correct the computed end-results thereby. The area of the aperture of the pupil and a correction for this are quite essential. Reeves [15] worked this out and gave a satisfactory logarithmic intensity curve. It is quite evident that as an area stimulated increases in relation to pupillary

14. Parsons, cited by Piéron, H.: De la variation de l'énergie liminaire en fonction de la durée d'excitation pour la vision fovéale et périphérique, Compt. rend. Acad. d. sc. **170**:1203, 1920; **178**:66, 1924; **181**:818, 1925. Parsons.[2]

15. Reeves, P.: The Effects of Various Physical Stimuli on Pupillary Area and Retinal Sensitivity, J. Franklin Inst. **184**:719, 1917; **186**:753, 1918.

diameter, this must be followed by an intensity increase in luminosity and an actual increase in the size of the retinal image in a much greater proportion than the increase in pupillary diameter. Derby [10a] and his co-workers were among the first to give this adequate attention. The accompanying table shows in the second column the absolute correction based on Reeves'[15] curve, and in the third column, the adjusted correction which is to be used. This correction is based on experience, and with this apparatus it is nearer the value actually found. These results

Corrections for Width of Pupil

Pupil, Mm.	Derby	Reeves	Spaeth
1.0	0.04	3.6	3.5
1.5	0.09	1.6	1.5
2.0	0.16	0.9	0.85
2.5	0.25	0.7	0.65
3.0	0.36	0.5	0.475
3.5	0.49	0.35	0.375
4.0	0.64	0.29	0.315
4.5	0.81	0.2	0.235
5.0	1.00	0.15	0.18
5.5	1.21	0.09	0.125
6.0	1.44	0.05	0.09
6.5	1.69	0.03	0.075
7.0	1.96	0.015	0.067
8.0	2.56	0.007	0.06
9.0	3.24	0.005	0.05

are modified by various factors such as light lost by dispersion, especially in the greater degrees of illumination, and applicable to the greater defects, that is, with larger pupils and an impaired light sense. The patient with a smaller pupil is more easily tested, and the probable error in his case is smaller. Last, the results show the necessity for fixing the pupil before testing with a mild miotic, such as a 0.5 per cent solution of pilocarpine. Various men, such as Hess and Lehmann,[16] have thought that miotics caused a decrease in the threshold of retinal response and the development of scotomas, by reason of their effect on the parasympathetic nervous system. The decrease, however, is a relative one, and I feel it is due only to the lessened degree of illumination on the retina

16. Hess, W. R., and Lehmann, F. E.: Der Einfluss vegetativer Reizstoffe auf den Adaptierungszustand der Retina, Arch. f. d. ges. Physiol. **211**:603, 1926.

because of the decreased area of the pupillary aperture. If the patient is not already under a mydriatic or a miotic, I use a 0.5 per cent solution of pilocarpine for fixation of the pupil. This again emphasizes the necessity for a corrective factor in this matter of determination of light threshold.

Preliminary light adaptation is essential to all testing. By establishing a standard for the patient, the greatest factor in variability of dark adaptation is eliminated. For, as Derby said, "the course of dark adaptation is obviously influenced by the level of illumination to which the eye has been influenced immediately before adaptation to darkness begins." The rate or progress in this adaptation need not influence results, in that the factors connected with this have been satisfactorily determined. Many pathologic and normal cases show a slower rate than others; hence

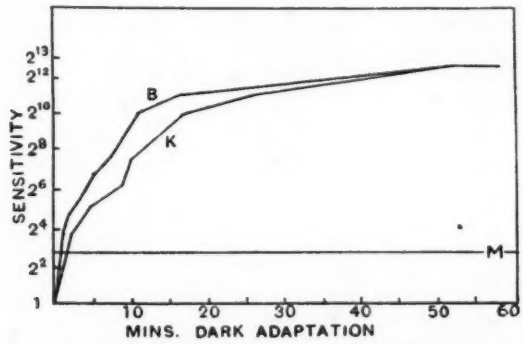

Fig. 6.—The increase of sensitivity during dark adaptation. *M* is the threshold of macular adaptation; *B* and *K*, different subjects. (Best: Ztschr. f. Biol. **68**: 119, 1917.)

an established time interval of twenty minutes was used as a routine. Many cases were again checked after an additional five to thirty minutes; such rare differences were shown that they were considered negligible for routine studies, but of importance in the isolated cases. Figure 6 is Best's [17] curve of dark adaptation and sensitivity rise as he presented it in 1917. *M* is the threshold for macular adaptation while *B* and *K* are two different subjects. It is interesting that Best was first in failing to find that lag in the early minutes of dark adaptation which Hecht found and described. Naturally, simultaneous bilateral adaptation both for preliminary light and for subsequent darkness is essential. The controversies relative to dark adaptation at the fovea can be disregarded. This is obvious when one glances at the chart illustrating the size and the

17. Best, F.: Dunkelanpassung des Auges mit Leuchtfarben, Ztschr. f. Biol. **68**:111, 1917-1918.

position of the retinal area stimulated. Central visual acuity in dark adaptation has been sufficiently covered by reference to the charts made after Evans and Breuer and Pertz. It is important to add here that care must be taken that light sense alone is being measured, and that nothing which savors of an epicritic nature enters into the methods used. The sole purpose of this paper is to present a method whereby findings relative to the minimum light threshold can be utilized clinically, and is not to discuss the detailed findings of foveal and peripheral adaptation and the numbers and kinds of recipient elements. While these are of tremendous importance in terms of retinal physiology, their consideration herein would add nothing to my topic. Kovács [18] has done a tremendous amount of work on this subject, and it is well worth one's time to read his studies in the original. A fair standardization of adaptation is approached by using the same interval under all circumstances. Those changes which appear after from thirty to seventy minutes of continued dark adaptation seem to be only of academic and incidental importance herein.

The method of presenting the results is rather important. All workers to date have expressed them in terms of microlamberts or photons or used some similar basis for the degree of luminosity. I have rather arbitrarily chosen a scale of one hundred parts for expressing my results. The greater the number, the more nearly perfect is the patient's light minimum; the lower the number, the greater is his light threshold, and hence his light sense defect; for example, from 1.5 to 2.5 is a very satisfactory light threshold, while from 0.01 to 0.001 would be a very high and pathologic light sense threshold. It seemed unwise to utilize micromillilamberts as a scale, for several reasons: The range of numbers is always large, and while more accurate, this scale gives no immediate information as to the degree of approach to normality in a given case. Further, it seems that a scale in microlamberts, and especially micromillilamberts, to be constant demands a most careful technic and unusual cooperation on the part of the patient. Even then the exact figures may be doubted. In these examinations it is not as important to know the exact amount of light the patient can identify as his light threshold as to know how nearly normal he may be as compared with others who apparently are free from ophthalmic pathologic processes, and also how great are the fluctuations in these readings. Further, by simple multiplication with a standard coefficient, the result found can be converted into any system of illumination desired. Regard-

18. Kovács, A.: Ueber den Einfluss der Dunkeladaptation auf die Empfindungszeit und den zeitlichen Verlauf der Gesichtsempfindung, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. (Abt. 2) **54**:161, 1922.

less of what method is used, no two persons can obtain exactly the same readings in a given patient, and the same observer will always find different readings at different times in the same patient even when all qualifying conditions are carefully matched.

The apparatus used in this study was sent to an electrical laboratory for microlambert calibration, but nothing further was done relative to this, for it was not thought necessary or desirable. The possibilities of error in calibration also were pointed out to me by this laboratory, and their work is of such a high standard that the opinion must be considered as valuable. It may be that I am in error relative to this opinion. If so, subsequent changes and corrections will be made. I feel, however, for the time being that the method in use is more applicable than others. There is no doubt that in such instruments as that presented by Derby and his co-workers, microlambert calibration and readings are not only best but are also necessary. This doubtful advantage for clinical application cannot be considered, though it is necessary for satisfactory and accurate research work. The last part of the aforementioned third group of factors includes methods of testing as to ease of operation and the value of the test from a clinical standpoint. The first of these subjects will be covered in the description of the instrument, and the last in the closing portions of this paper.

APPARATUS FOR DETERMINATION OF LIGHT THRESHOLD

The instrument, made by Wall and Ochs of Philadelphia, is quite simple (figs. 7, 8 and 9). The goggles for dark adaptation are arranged with slides and windows so that they are light proof at all times, even when the prism carrier is in place. The special value of the entire apparatus lies in the fact that everything may be carried out in an ophthalmologist's ordinary dark room, even with the usual illumination of the room and without the necessity of wasting time for self dark adaptation. While the patient's dark adaptation is developing, it is my practice to have him wait in the usual office reception room and, as the time limit approaches, to move him to the dark room for the remainder of the examination. The preliminary light adaptation is done with an ordinary blue glass "day-light" equivalent globe from a distance of 9 feet (2.7 meters) so that at the eye the illumination is approximately 5 foot-candles. The light source is a 5 foot-candle luminosity obtained with an ordinary seasoned flashlight bulb capped with a plus 20 sphere and with a frosted lens cap so held as to touch the window in the prism carrier. This degree of illumination has been checked at various times by Feldman[19] with his photo-electric-galvanometer photometer, and

19. Feldman, J. B.: An Improved Illuminator and Pupillometer, Arch. Ophth. 9:974 (June) 1933.

these globes have shown a full hundred hours of usableness before any change has appeared in the amount of their illumination, the same rheostatic stop being used at all times. In certain cases a light threshold may be so high, because of pathologic changes, that it will be necessary to step up the rheostatic control to obtain greater illumination through the aperture. When this is done it is necessary to know the difference in

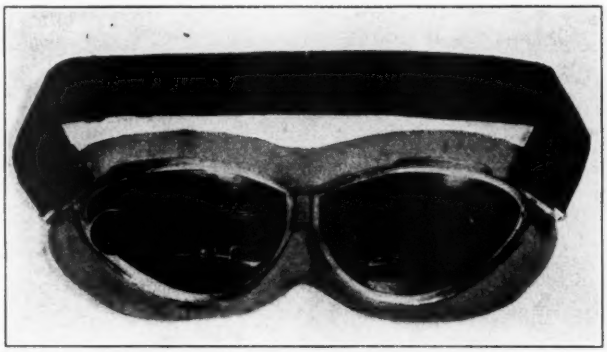

Fig. 7.—Dark adaptation goggles. One slide is open; the other is closed. The slide is opened after the prism carrier has been placed on a lug and pin in front of an aperture.

Fig. 8.—Prism carrier in place, with slide behind it opened for the threshold determination.

illumination between the point on the rheostat ordinarily used and that which is now being delivered to the lamp by means of the increased voltage. If the ordinary amount from the lamp, 5 foot-candles, has been increased temporarily to, say, 7 foot-candles, the light threshold must be stepped up in the same proportion; i. e., in this instance it would be 0.72 higher than that shown by the table of corrections. In this manner the illumination used and the amount of light seen can be changed and controlled at will as long as the initial and modified candle-powers are known. The rheostat is an ordinary plug-in wall rheostat as manufac-

tured by various firms. In this instance the Westinghouse electric rheostat was selected because it was conveniently at hand. The prism carrier has a small perforated lug for secure attachment to the goggles while in use. The two prisms are inclosed in a case from base to apex, their flat surfaces parallel and in approximation, their oblique surfaces being outermost. As these prisms are separated, the thickness of glass in front of the aperture is proportionately decreased. The size of the retinal image, as obtained through the five perforations, has been discussed.

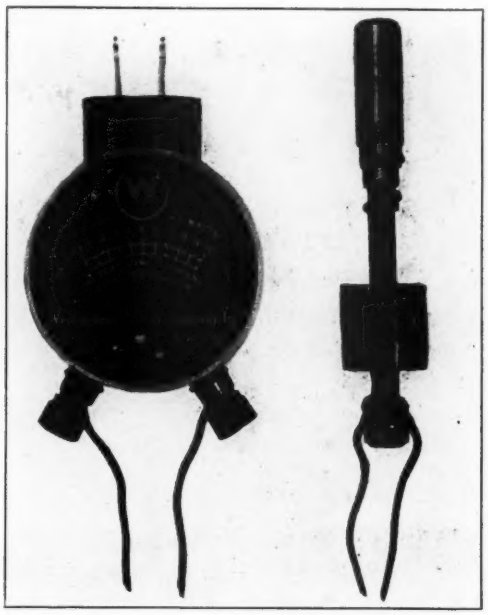

Fig. 9.—Light source and rheostat.

The glass used was made by the Corning Glass Company. It was selected after the trial of several different samples. Recently, Duke-Elder [8] called attention to the fact that the sensitivity curves for the various wavelengths show considerable differences. The value for green is always the highest. This applies not only to the consideration of the colors, yellow, blue and red (in that order of decrease), but also to pure achromatic white light. Wentworth [20] obtained measurements in the completely dark-adapted eye which showed conclusively that a measurement for maximum sensitivity must be made with green light of a certain

20. Wentworth, H. A.: Quantitative Study of Achromatic and Chromatic Sensitivity from the Center to the Periphery of the Visual Field, Psychol. Monog. **40**:1, 1930.

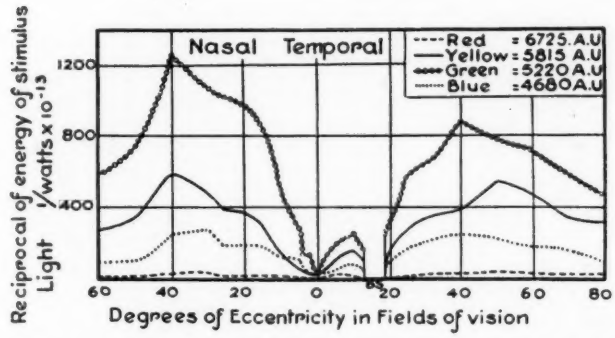

Fig. 10.—The achromatic sensitivity of the retina to spectral lights from the center to the periphery of the field in the horizontal meridian expressed in terms of energy (dark adaptation). (After Wentworth.)

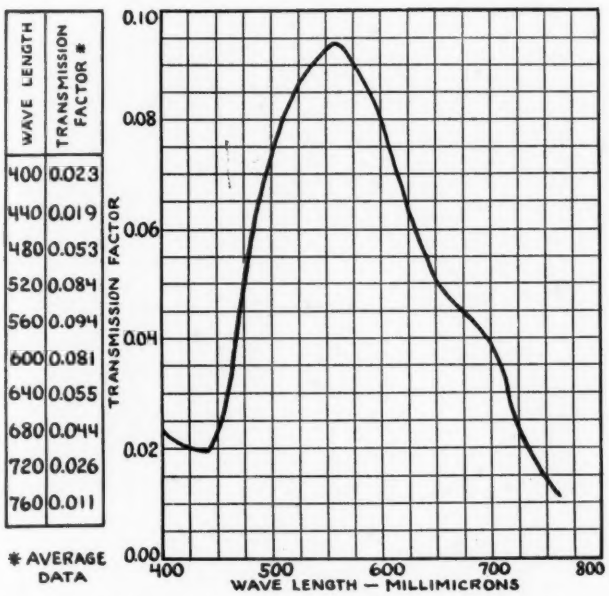

Fig. 11.—Spectrophotometric curve of prisms, and transmission factor of glass used for wedges. Measurements were made with a photo-electric cell spectrophotometer. The results are dependable to $\pm 0.00_5$ unit. The thickness of the glass is 0.49 mm.

fixed spectral wavelength. Figure 10 illustrates this well. According to Wentworth, the determination of the absolute light minimum should be made in the completely dark-adapted eye with green light and at the most sensitive point on the retina, i. e., 40 degrees in the nasal quadrant. For many reasons this is impracticable, if one desires to add this procedure to one's clinical armamentarium; hence, the deliberate selection of the parafoveal region for examination rather than the more sensitive but less accessible nasal periphery (Nagel and Schaffer).[21] Figure 11 is the spectrophotometric curve of the glass used in these prisms, examination and plotting having been done at my request by the Electric Testing Laboratories of New York. Maximum transmission occurs between wavelengths 520 and 600 millimicrons. In the visible spectrum this ranges from the blue green to the green yellow. The selection of glass with this spectral transmission is most logical and seems sufficiently evident for the apparatus under consideration.

The instrument is by no means an accurate laboratory research machine—its limitations are definite—but it does furnish a constant means of conveniently obtaining valuable data. Purposely nothing has been said of the various other adaptometers, photometric systems, glasses, disks and lanterns which have appeared since the first published and valuable investigations of Arago [22] in 1858. They all have certain limitations, as does the instrument just described, but at least one can obtain conveniently with the latter something which, I feel, is of inestimable value in diagnostic and prognostic procedures—something which has been needlessly neglected because of the inaccuracies and the mechanical defects and difficulties which have been too often connected with the examination of the light minimum in the dark-adapted eye.

CLINICAL FINDINGS

Data on pathologic conditions in a large number of persons examined in the past five years are presented here as a preliminary report. The majority of patients were normal, as would be expected. The data illustrate the possibilities of any adaptometer. (A certain number of patients were also examined with the Birch-Hirschfeld wedge photometer.[23]) The types of cases in which an examination (and in some

21. Nagel, W. A., and Schaffer, K. L.: Ueber das Verhalten der Netzhautzapfen bei Dunkeladaptation des Auges, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **34**:271, 1904; **35**:88, 1905.

22. Arago: Œuvres complètes, Paris, 1858, p. 10.

23. Birch-Hirschfeld: Hemeralopia, in Axenfeld, K. T.: Lehrbuch der Augenheilkunde, Jena, Gustav Fischer, 1910, vol. 5, p. 135.

instances a reexamination) was made during this period were most varied. An incomplete list follows:

Glaucoma, all types, diagnosis established	30
Glaucoma suspected (preglaucomas)	11
Retinal detachment (all degrees and types)	7
Retinitis pigmentosa	5
Cataract (all types)	31
Keratitis, chronic, with corneal cicatrices	7
Papillitis	5
Papilledema, but with some visual acuity	10
Atrophy of optic nerve	
Primary	10
Secondary (posttraumatic)	4
Postneuritic (consecutive)	17
Chorioretinitis, diabetic and nephritic	29
Amblyopia with squint and in many cases with demonstrable central scotoma	11
Central macular chorioretinitis	6
Albinism, congenital amblyopia and ocular nystagmus with high degree of amblyopia	4
Retrobulbar neuritis, multiple sclerosis, toxic amblyopia	6

In all types of glaucoma there was a positive increase in the light threshold. This was greatest in the acute and subacute forms, at times being so marked that the limits of the prisms were exhausted. So-called acute inflammatory fulminating glaucoma showed the greatest impairment in the light threshold, and the degree of this impairment was roughly proportionate to the amount of visual acuity present after operation. In two cases, it seemed, however, as if the light minimum was less impaired than the central acuity. In one such case, that of a very acute secondary iritic glaucoma, the visual acuity was 6/60 and the light minimum 0.60. The patient recovered with 6/5 vision and with a return of the light threshold to normal. This same relationship was seen in chronic simple glaucoma of long standing, even when the fields were still of fair proportions. Figure 4 shows this to some extent. In secondary glaucoma, as with swelling of the lens, there were changes which could have been due to the opacity of the lens, for there was little difference between the light minimum in this type of case and that in an uncomplicated cataract of equal degree. Persons with secondary glaucoma from other causes showed, after some time, an increase in the light minimum, but it was rather less than that in persons with primary glaucoma, considering the ratio between the visual acuity and the light minimum. Patients with noninflammatory glaucoma, in whom there was a great fall in visual acuity, as from 2/60 to 6/60 central vision, showed in some instances a light sense minimum so high that it could be determined with these prisms only inaccurately and after an

increase in the basic illumination. The prognosis in these cases was properly considered bad.

A preglaucomatous syndrome, if such a symptom complex is ever worked out, will certainly include positive indications of an early increase in the light minimum. For the time being, the damage to the light sense must be considered as a sequela of increased tension (for want of positive evidence based on vascular, toxic or other causes). It seems likely that the early damage is from recurrent though perhaps transient periods of uptension, at a time when the diagnosis is still unthought of and long before other symptoms have presented themselves. In eleven such cases, two of the patients have come to operation, and the diagnosis is almost certain in several others. In these cases an increase in tension was not found, not because it was never present, but more likely because the examination was made at a time or times when the tension had temporarily dropped to normal or to the upper limits of normal. It is impossible to confirm this statement, for the opportunity has not as yet appeared, but I feel certain that time will prove the correctness of the following conjecture—that an increase in the light minimum antedates losses in central visual acuity and field and that it appears before ophthalmoscopic cupping can be seen and should be of such valuable diagnostic import that treatment, if instituted immediately, will save the patient from later disability because of the ravages of this condition. It is interesting and relevant to mention here that Evans' work on angioscotometry, which also utilizes a light threshold test, is along these same lines and has a similar aim.

All cases of retinal detachment showed such a marked increase in the minimum light sense that one may consider them as largely choroiditic, and assume as well that the great impairment was due in a large part to selective damage to the rods, the cones and the pigmented epithelium by reason of the detachment. Recovery from the detachment, as measured by a reattachment of the retina and a return of some portion of the field and of the central visual acuity, did not result in a proportionate improvement in the minimum light sense.[24] Several authors,

24. Since the presentation of this paper, Kronfeld has published a paper on the function of the reattached retina (Arch. Ophth. **10**:646 [Nov.] 1933). His paper had been submitted for publication after presentation early in this year, but its contents were unknown to me until it was published; so priority for the aforementioned finding, to which this footnote applies, belongs to him. It is interesting, however, to observe that both of us have separately confirmed such a permanent loss in retinal dyscritic function. This most significant statement is quoted verbatim from his paper: "From these observations it seems as though the differential threshold for intensities of light is considerably higher for a reattached than for a normal retina." There are other varied factors in his work relative to the physiology of the reattached retina, but this confirmation may be properly included herein.

Evans,[6] Gasteiger,[25] and others, have already pointed out the marked fall in sensitivity found in all forms of retinal edema, as in detachment, albuminuric edema and the postural retinal edema which accompanies arterial hypotension.

Retinitis pigmentosa was always accompanied by a great increase in the light minimum. Patients with great impairment in acuity had an even greater impairment of their light minimum. Two patients especially, each with better than 6/9 vision, had an impairment of the light threshold of less than one tenth of the normal. In one patient, acuity had been maintained fairly well, but the light minimum had been rising steadily over a period of three years. Within a few months preceding the last examination the central acuity had fallen abruptly from better than 6/7.5 to less than 6/12, and in addition the patient was complaining of considerable metamorphopsia in the affected eye. One patient with a probable central visual acuity of 1/150 had a light threshold so markedly impaired that measurement was possible only with greatly increased illumination.

In both keratitis with permanent corneal maculae and in cataract of all varieties, except complicated cataract of high degree, the light sense minimum was less impaired than visual acuity.

Patients with papillitis showed a lower degree of impairment in the light sense minimum than in central visual acuity. This, however, was early in the course of the optic neuritis. Later in the course of some of the cases the impairment in the light sense minimum became greater than the fall in central visual acuity. So far as papilledema was concerned, there was little if any upset in the ratio between the fall in acuity and the rise in the light sense minimum—except in cases of marked defects in the visual field. In such instances the greater the defect, the greater was the rise in the light sense threshold. It seems this can be fairly well accounted for by reason of other statements made earlier herein.

The optic atrophies followed a fairly consistent grouping. They all showed a definite impairment in the light minimum, but were dissimilar in respect to the ratio between this and the fall in central visual acuity. In tabetic or primary atrophy of the optic nerve it was found that the impairment of the light minimum was greater than the fall in central visual acuity, though in well established postneuritic atrophy the permanent ratio found was at times almost as low as was that usually found in primary atrophy. It is quite likely that the various conventional defects in the visual field in these conditions have much to do with these findings. Secondary optic atrophy gave in all instances a better value

25. Gasteiger, H.: Ueber Störungen der Dunkeladaptation bei Sehnervenerkrankungen und ihre diagnostische Verwertbarkeit, Klin. Monatsbl. f. Augenh. **78**:827, 1927.

for light sense threshold than was found for the percentage of central visual acuity still present. Of course, in these cases defects in the visual field, and especially central, absolute and relative scotomas, confused to some extent the mechanics of the tests. One posttraumatic case, however, following a hemorrhage into the sheath of the optic nerve, did show at one time a light sense minimum of 0.40 when visual acuity was less than 6/150. It is interesting that vision in this case improved to 6/6.

The various types of retinitis and chorioretinitis showed many interesting variations. In most instances the patients with a high degree of choroidal disease showed the greatest defect in the light threshold. Many nephritic patients with great edema and with massive exudates showed great impairment—far greater in degree than the loss of central acuity. Patients with massive macular pathologic processes, tuberculous and traumatic, showed plainly the influence this paracentral pathologic process has on the parafoveal light threshold. Patients with hemorrhagic changes, even massive, as seen in two cases of vascular branch thrombosis (not the central retinal vein), showed relatively little change from the normal. One patient with a diabetic subretinal hemorrhage of high degree showed a fair light threshold, at least one that was definitely determinable long after visual acuity had become so low as to be indefinable.

The cases of amblyopia ex anopsia with central scotoma and with squint were universally interesting. All patients with this condition had perhaps some slight impairment, but it was most uncommon to find one in whom the light minimum was as low in proportion as the visual acuity. As Evans [6] pointed out, the absolute scotoma of strabismus occupies an area about 1 degree in radius, and since this same distance eccentric from the fovea has a light sensitivity a thousand times greater than that of the fovea itself, and as that portion of the retina which is best adapted for a position under observation is bound to be used for eccentric fixation, it follows that squinting eyes (fixing with an eccentric region of the retina) can be expected to have a light sense threshold which is lower (that is, more nearly normal) than the percentage of visual acuity, or perhaps even better than that of the normal eye. Visual acuity 1 degree from the macula has already dropped to 6/12, while the same region has at the same time risen appreciably in sensitivity under dark adaptation. The following case is rather interesting and not uncommon: An 11 year old child, with a central scotoma and monolateral convergent strabismus, with a corrected vision of less than 6/30 in the right eye, and 6/5 in the left eye, had a light minimum of 0.80 in the right eye and from 0.89 to 0.90 in the left eye. This may account in part for the satisfactory results which are obtainable in many of these cases

through proper orthoptic exercises. I do not believe that eccentric fixation accounts wholly for the changes in the light sense in these cases.

All but one of the patients with albinism, a high degree of ocular nystagmus and the various other congenital retinal and oculomotor disturbances had as great a proportionate increase (if not greater) in the light threshold as a decrease in the central visual acuity. There were but four cases in this series, so one cannot consider these findings significant.

The group of cases of retrobulbar neuritis was also interesting. In these, the light sense minimum was always more impaired than the central visual acuity. All the patients, however, showed central scotomas, and one with a cecocentral scotoma rather larger than usual had the disturbing inconsistency of showing at one stage a light minimum disproportionately lower (better) than the visual acuity, though when vision had recovered from less than 2/60 to 6/12 the decrease in acuity was relatively less than the permanent impairment in the light minimum.

It is well to mention here briefly some data on the light threshold in pathologic conditions of the eye as found by other investigators. The list naturally is incomplete but interesting and relevant. Von Hess,[26] Hübener,[27] Nakamura [28] and others found marked changes in nyctalopia, in hemeralopia and in other instances of avitaminosis. König and Dieterici [29] and Hecht [12] thought that absolute color blindness includes a central scotoma, and that it is a point in favor of the theory of predominance of rods in dark adaptation and visual acuity. Förester [30] and Lohmann [31] found a decrease in the capacity for dark adaptation in various forms of choroiditis and retinitis, in glaucoma and in optic neuritis and optic atrophy. Stargardt [32] found an absolute lack of capacity for dark adaptation in retinal detachment. Lohmann [31] found normal adaptation in pure macular defects as in Haab's macular degeneration.

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26. von Hess: Untersuchungen über das Sehen und über die Pupillenreaktion, Arch. f. Augenh. **59**:142, 1908.

27. Hübener: Untersuchungen über hemeralopische Beschwerden und Adaptation bei Soldaten, Arch. f. Ophth. **93**:360, 1917.

28. Nakamura, B.: Dark Adaptation with Homonymous Hemeralopia, Klin. Monatsbl. f. Augenh. **69**:320, 1921; Light Sense in Various Forms of Night Blindness: Oguchi's Disease and Mizuo's Symptom, ibid. **69**:145, 1921.

29. König, A., and Dieterici, C.: Die Grundempfindungen in normalen und anomalen Farbensystemen und ihre Intensitätsverteilung im Spektrum, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **4**:241, 1884.

30. Förester: Lichtsinn bei Krankheiten der Choroidea und Retina, Klin. Monatsbl. f. Augenh. **9**:337, 1873.

31. Lohmann, W.: Untersuchungen über Adaptation und ihre Bedeutung für Erkrankungen des Augenhintergrundes, Arch. f. Ophth. **65**:365, 1907.

32. Stargardt, K.: Ueber Störungen der Dunkeladaptation, Arch. f. Ophth. **73**:77, 110. Lohmann.[31]

Jess [33] found a disturbance in dark adaptation in a normal eye when the other was severely inflamed with its light sense threshold markedly impaired. Schindler [34] and Behr [35] found definite disturbances in optic neuritis, but the light sense threshold was normal in pure papilledema (choked disk). They emphasized the importance that this test could have in the differentiation of true and pseudo-optic neuritis. Rutgers [36] wrote extensively of the disturbance of dark adaptation in congenital pigmentary degenerations. Also he [37] found less disturbance in the tabetic type of atrophy than in other forms. The work of Derby, Waite and Kirk [38] relative to glaucoma is classic. Kleczkowski [39] found a ring-shaped parafoveal scotoma in dark adaptation in certain cases of congenital amblyopia. Feigenbaum [40] studied the effect of exposure to light and darkness on the intra-ocular pressure of normal and glaucomatous patients and also investigated their course of dark adaptation with and without the influence of pilocarpine. He feels that the different types of glaucoma show characteristic variations in intra-ocular pressure. Also he stated that disturbances of dark adaptation in these cases is an early clinical sign. Many more instances of this type of work can be selected, but these suffice for the illustration desired.

I am well aware of the fact that my series is wofully small for any authoritative statistics; still it shows that a determination of the light threshold is not wholly a waste of time. But little attempt has been made to explain the findings presented. Further work must be done in a great many similar cases to obtain satisfactory consistent findings. At that time I may feel able to explain the various results.

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33. Jess, A.: Ueber Adaptationsstörungen auf sympathischen Wege sowie Demonstration von Gesichtsfeldern bei erworbener Hemeralopie, Ber. d. Heidelberg. ophth. Gesellsch., Aug., 1918, p. 217.

34. Schindler: Dark Adaptation, Klin. Monatsbl. f. Augenh. **68**:710, 1922.

35. Behr: Das Verhalten und die diagnostische Bedeutung der Dunkeladaptation bei den verschiedenen Erkrankungen des Sehnervenstammes: I, Klin. Monatsbl. f. Augenh. **55**:193, 1910; II, **55**:449, 1910.

36. Rutgers, G. E.: Dark Adaptation in Some Eye Diseases, Klin. Monatsbl. f. Augenh. **71**:449, 1923.

37. Rutgers, G. E.: Dark Adaptation in Optic Nerve Atrophy, Monatsbl. f. Augenh. **72**:8, 1924.

38. Derby, Waite, and Kirk (footnotes 10 *b* and *c*).

39. von Kleczkowski, T.: Dark Adaptation of the Eyes, Arch. f. Augenh. **88**: 253, 1921.

40. Feigenbaum, A.: Ueber vorübergehende und dauernde Störungen der Dunkeladaptation beim Glaukom, Klin. Monatsbl. f. Augenh. **80**:596, 1928; Ueber den Einfluss der Belichtungen und Verdunkelung auf den intra-okularen Druck normaler und glaucomatöser Augen, ibid. **80**:577, 1928.

The surface of the subject is barely scratched. If for no other reason than this, a plea is made here for the development of standard instruments and technic for the routine study of the light minimum or, better, the light sense threshold, which should be considered a valuable procedure in the practice of ophthalmology. It is one which is neglected needlessly and with a casualness which is too evidently due to lack of concern and interest.

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BIASTIGMATISM

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Some have been alarmed at the papers read at recent international congresses on biastigmatism and a suggested triastigmatism, and at the complicated way of discovering and treating such conditions. It is known by all that astigmatism is usually due to an unequal curvature of the anterior surface of the cornea, and that this astigmatism may be masked or increased by an unusual curvature of the posterior corneal surface or of either or both surfaces of the lens, or by a partial dislocation or tilting of the lens.

These difficulties arise only when one trusts to the readings of the ophthalmometer for finding the power and the axis of the correcting cylinder. When ordering glasses, one wishes to find the total astigmatism of the eye and the cylinder that will correct it when placed at the assigned distance (say, 13.6 mm.) from the cornea. Retinoscopy when carefully done will determine not only the power of the required cylinder but also the exact angle at which its plane axis should be placed.

The power C of a cylinder set at axis α has the power $C \sin^2 \alpha$ in the horizontal meridian, and the power $C \cos^2 \alpha$ in the vertical meridian. The experiment may be easily made with a —4 D. cyl. and a lens measure. The power is $-4 \sin^2 0 = 0$ in the plane axis (at angle 0 degrees); at an angle of 30 degrees the power is $-4 \sin^2 30$ degrees, or $-4 \left(\frac{1}{2}\right)^2 = -1$D.; at 60 degrees the power is $-4 \left(\frac{\sqrt{3}}{2}\right)^2 = -3$D., and at 90 degrees the power is $-4 \sin^2 90$ degrees or $-4 \cos^2 0$ degrees $= -4$D.

The first point to be recognized is that two cylinders set at any angle to each other may be replaced by a spherical lens and by one cylinder set at an angle ϕ which can be found.

If a cylinder of power C_1 is set at an angle α to the horizontal and a cylinder of power C_2 is set at an angle β, the combined effect of the two cylinders is that of a spherical lens of power D and a cylinder of power C′ set at an angle ϕ, where

$$C_1 \sin^2 \alpha + C_2 \sin^2 \beta = D + C' \sin^2 \phi$$

or

$$\frac{C_1}{2}(1 - \cos 2\alpha) + {}_2 \quad (1 - \cos 2\beta) = D + \frac{C'}{4}(1 - \cos 2\phi)$$

$$\therefore 2D = C_1 + C_2 - C'$$

and

$$C_1 \cos 2\alpha + C_2 \cos 2\beta = C' \cos 2\phi$$

It remains to determine the value of C′ and ϕ.

However, the combination of the two cylinders C_1 and C_2 is rotated, as long as the angle $(\beta - \alpha)$ between them remains constant, the power of the combination will be the same, i. e., D and C′ will be constant, and it is only α, β and ϕ that will vary. In other words, any arbitrary angle θ may be taken as the zero angle from which α, β and ϕ are measured. Hence

$$C_1 \cos 2(\alpha - \theta) + C_2 \cos 2(\beta - \theta) = C' \cos 2(\phi - \theta)$$

But as $\cos 2(A - B) = \sin 2A \sin 2B + \cos 2A \cos 2B$, one may rewrite the foregoing expression in the form,

$$\sin 2\theta (C_1 \sin 2\alpha + C_2 \sin 2\beta - C' \sin 2\phi) + \cos 2\theta (C_1 \cos 2\alpha + C_2 \cos 2\beta - C' \cos 2\phi) = 0$$

Now as θ is entirely arbitrary, it is clear that each expression in brackets must be of zero value.

$$\therefore C_1 \sin 2\alpha + C_2 \sin 2\beta = C' \sin 2\phi$$
$$C_1 \cos 2\alpha + C_2 \cos 2\beta = C' \cos 2\phi$$

$$\text{so } \frac{C_1 \sin 2\alpha + C_2 \sin 2\beta}{C_1 \cos 2\alpha + C_2 \cos 2\beta} = \frac{C' \sin 2\phi}{C' \cos 2\phi} = \tan 2\phi$$

and

$$C' = \frac{C_1 \sin 2\alpha + C_2 \sin 2\beta}{\sin 2\phi} \text{ or } \frac{C_1 \cos 2\alpha + C_2 \cos 2\beta}{\cos 2\phi}$$

whichever is the more convenient.

It is an easy matter to add a third cylinder to this result; one has merely to add C_3 set at an angle γ to C′ set at ϕ:

$$\frac{C' \sin 2\phi + C_3 \sin 2\psi}{C' \cos 2\phi + C_3 \cos 2\psi} = \frac{C'' \sin 2\phi}{C'' \cos 2\phi} = \tan 2\psi$$

and

$$C' + C_3 = 2D' + C'', \text{ but } C' = C_1 + C_2 - 2D$$
$$\therefore C_1 + C_2 + C_3 = 2(D + D') + C''$$

C″ is, of course, set at angle ψ which is determined as before.

One may therefore consider that the total astigmatism of the eye is represented by C_2 set at angle β in the spectacle plane; in retinoscopy one must find a glass of $-C_2$ in power set at this angle to correct it. It will be found that the nearer in power and the nearer in position the correcting cylinder is placed during a retinoscopy, the greater is the magnification of the error in position of the cylinder. This magnification, which is an insuperable objection in the use of cylinders to correct the amount of astigmatism present by retinoscopy, is of enormous advantage in determining the exact position of the cylinder.

May I be pardoned for giving in detail the procedure which I adopt in doing a retinoscopy, e. g., in a case of compound myopic astigmatism? I personally prefer the concave mirror (1) because it gives the best illumination, (2) because one sees lenticular opacities less clearly and (3) because one is less troubled with the swirling shadows that some-

times impair the accuracy of one's results. The patient, whose pupils have been dilated and whose accommodation has been temporarily paralyzed by homatropine, is directed at first to look past my head, at the wall opposite, while I seat myself at first about 80 cm. from his eyes for convenience in altering lenses in the trial frame. I note that on swaying the mirror from side to side the reflex light is fairly bright and the movement of the light is in the same direction as the mirror and fairly rapid; on tilting the mirror in the vertical direction, the reflex light is much less bright and the movement is much slower. I conclude that the eye is myopic in the horizontal direction about —2 or —3 D., and still more myopic in the vertical direction. One movement of the mirror is enough; nothing is gained by repeating it. I try a —2.5 D. lens in the trial frame and find that the movement of the reflex light is much more rapid; when I lean back I find, on slowly swaying the mirror, that I can see the light moving still more rapidly with the mirror. I now seat myself at exactly 1 meter from the spectacle plane, and tell the patient to look directly at the mirror. I insert a —2.75 D. lens in the trial frame. The reflex is now very bright, and although I move the mirror slowly, I cannot tell which way it moved; the light suddenly disappears. On leaning backward, I find that the light moves very rapidly in the same direction as the mirror, but on leaning forward the light moves against the mirror, so I know that the eye is exactly corrected in this meridian for 1 meter, and that in its present state it will be corrected for distance by —3.75 D. in this meridian. By moving backward 13 inches (33 cm.) I can find the point of reversal at 133 cm. (—0.75 D.), and on leaning forward about 8 inches (20 cm.) the point of reversal will be at 80 cm. (—1.25 D.).

Now for the other meridian: Leaving the —2.75 D. in position, I add another —2.5 D. spherical lens to it, and tilt the mirror in about the vertical meridian; the light moves much more rapidly in the same direction, and I find that the point of reversal is reached with a —3 D. lens at 1 meter. One now has found the power of the spherical lens for distance in the present state of the eye (—3.75 D. sph.) and the power of the cylinder (—3 D. cyl.), but the angle of the axis is required.

I take out the —3 D. lens and substitute a weaker cylinder than that required; I put in a —2 D. cyl. with its plane axis horizontal and then tilt the mirror along its working axis, i. e., in the vertical meridian; the light moves obliquely in the 115 degree meridian and sheers off below. If the error were given exactly, the cylinder should be rotated until its plane axis was in the 25 degree meridian; knowing, however, that the error is magnified, I rotate it upward about 8 degrees, i.e., about one third of the suggested error and tilt the mirror again at right angles

to the plane axis. This is quite easy to do, as one can see which is the plane axis by the ground glass line on the cylinder. The light again moves, still obliquely with the mirror, so the cylinder requires raising again a trifle; finally, when the axis is put in the 10 degree meridian, and when the mirror is tilted in the direction of the working axis of the cylinder, it will be found that the light moves exactly in the plane of the working meridian, and this is the true axis of the cylinder. The correction for distance in the present condition of the eye is — 3.75 D. sph. — 3 D. cyl. ax. 10 degrees.

The power of the cylinder must always be found by retinoscopy with spherical lenses, and the axis always by a weaker cylinder, say, two-thirds or one-half the real power, in this way.

In this case the actual refraction of the eye for 1 meter in the spectacle plane is regarded as + 2.75 D. sph. + 3 D. cyl. ax. 10 degrees, requiring, of course a similar glass, but of negative power to correct it. Consequently, in my first trial when I put in a — 2 D. cyl. ax. 0 degree, the equation for tan 2 ϕ will be

$$\frac{-2 \sin 0^\circ + 3 \sin 20^\circ}{-2 \cos 0^\circ + 3 \cos 20^\circ} = \frac{1.02606}{0.81908} = 1.2513 = \tan 2\phi$$

$$\therefore 2\phi = 51^\circ\ 22 \text{ or } \phi = 25^\circ\ 41$$

The true value of ϕ is 10 degrees, so the magnification of the error is about 2½; if 8 degrees is substituted for 0 degrees, the error is only 2 degrees but the magnification of the error is nearly 7: Hence the true axis is found by a very slight alteration of the position of the cylinder when the power of the cylinder is distinctly less than that required to correct the astigmatism of the eye.

In what form should such a lens as — 3.75 D. sph. — 3 D. cyl. ax. 10 degrees be prescribed? The eye ranges from side to side far more frequently than up and down, so a minus cylinder with its plane axis nearly horizontal will not add to its periscopic effect in this direction; consequently, it will be advisable to place the cylinder anteriorly and the concave spherical surface next the eye. If the patient expressed a wish for periscopic spectacles, a simple and efficient form would be + 3.75 D. cyl. ax. 90 ⌣ —6.75 D. sph. with the concave spherical surface next to the eye.

It will be inferred, I hope, from this note that the ophthalmometer is not a trustworthy guide to the discovery of the total astigmatism of the eye.

PERISCOPIC SPECTACLES

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Periscopic lenses are still regarded frequently as lenses which give good definition in the peripheral parts of the field. This is what is required in a photographic lens, and excellent photographic lenses have long been made; striking photographs are often given of test types placed 30 degrees from the axis of a biconvex lens, and these are contrasted with those that have been produced by a so-called periscopic lens of the same power. But this lens is not a periscopic spectacle lens; it is only a photographic lens provided with a fixed stop. The periscopic spectacle lens has a movable stop (the iris) and should give a well defined macular image of the eccentric part of the field under examination, when the eye ranges from side to side.

The diameter of a macular cone is about 0.002 mm., and for distinct vision, therefore, the radius of the circle of least confusion should be less than 0.001 mm. M. Dor has taught that 5 degrees from the fixation point the visual acuteness has diminished to 0.25 and at 10 degrees from the fixation point to 0.067 of that at the macula. To the eye it is a matter of entire indifference whether the images that are formed on the peripheral parts of the retina are accurately focused or not. They cannot be distinguished in any case. If the eye is fixed on one letter of a long word on this page, the fourth letter on either side of it cannot be recognized.

A patent specification of the American Optical Company of Southbridge, Mass., U. S. A. (262,319), entitled "Improvements in Ophthalmic Lenses," has been sent to me for my opinion. I hear that these lenses are now widely used, but from the specification, over 550 lines in length, I find that I cannot recommend the lenses. Without doubt they are better than the biconvex or biconcave form, but the principle of the method is unsound.

As suggested previously, the only satisfactory way of constructing a periscopic spectacle lens of a given power is to find, if possible, such two surfaces as will give rise to a circle of confusion, the area of which shall not be greater than the tip of a macular cone when the eye is rotated 30 degrees from the middle line. This is a most tedious business, and the short graphic method used for the "new lenses" is not satisfactory, as will be shown later. The specification does not state whether Ostwalt's, Tscherning's or Gullstrand's formulas and constants are used; the circle of confusion is not even mentioned, nor is the index of refraction given. For these reasons I am unable to criticize the mathematical work, but I have examined the two examples given, which are presumably among the best, and these are the results:

In the specification "the best possible lens for variations of both focus and astigmatism" of a — 8 D. lens is one with an ocular curve of — 10.40 D.

Using Gullstrand's constants, the standard index of refraction (1.523) and the axial thickness of 1 mm., I find that the area of the confusion circle is 1.36 times that of the tip of a macular cone, although the difference between the power of the lens for centric and eccentric refraction is only 0.065 D.

For the spherocylinder — 5 D. sph. — 3 D. cyl. the Southbridge Optical Company advise an anterior curve of + 2.75 D. and an ocular toric curve of — 7.75 D. and — 10.75 D. This is said to give "a maximum variation at any point of ¼ D. focal variation and ¼ D. astigmatic variation, which will give the maximum visual efficiency in both meridians of that power."

I find that in the — 5 D. meridian the eccentric power is — 5.4 D., and the circle of least confusion has an area of more than fifteen times that of the tip of a cone, so this is far from being a periscopic lens. In the — 8 D. meridian, the result is very good; the eccentric power is — 7.85 D., and the confusion circle is far smaller than that of the tip of a cone.

No warning is given about the thickness of a high convex periscopic lens with its extreme convexity on its anterior surface; this, as is well known, increases the effective back focal power greatly; e. g., a periscopic lens of ocular curvature — 7 D. and anterior curvature of + 17 D. (thickness, 5 mm.) will have an effective back focal power of over + 11 D., not + 10 D.

Again no mention is made of the difference in shape of a periscopic lens required for reading from that required for distance. For instance, if a + 6 D. periscopic lens is required for reading, its ocular surface should be about — 4.5 D.; but if the same power is required for distance, its ocular surface should be about — 7 D.

An accurate periscopic lens of — 8 D. would be one with an ocular curve of — 10.755 D. and an anterior curve of + 2.75 D.; this would give a centric power of — 8 D. and an eccentric power of — 7.88 D., and the confusion circle area would be only 0.0188 of that of the tip of a cone.

The only satisfactory way of making a toric periscopic lens for a concave spherocylinder, if the cylinder is over —1.5 D., is to make both anterior and ocular surfaces toric curves. For the given case, I would suggest $\frac{+ 2.75 \text{ D.} \quad + 4.2 \text{ D.}}{- 9.2116 \text{ D.} \quad - 10.755 \text{ D.}}$, or nearly this form.

For the — 5 D. meridian (+ 4.2 D. — 9.2116 D.), the central back focal power will be exactly — 5 D., the similar eccentric power will be — 4.93 D., and the area of the confusion circle will be only 0.1 of that of the tip of a cone. This is clearly unnecessarily exact.

OPTIC NEUROMYELITIS

REPORT OF TWO CASES

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Optic neuromyelitis is a rare disease, known for only twenty-five years. A little over fifty cases are cited in the literature, only five of which are recorded in English. The first anatomic-pathologic description was given in 1889 by Achard and Guineau, although a few cases were reported previously by other authors. Devich, in 1894, gave the disease its name; other cases were afterward published by Katz, Brissaud and Rochon-Duvigneaud and by Hilion, who in his thesis reported forty-five cases. Complete histopathologic examinations were recorded in sixteen of the twenty-four fatal cases. Later came the observations of Hoffman, Cobledick, Miashita, Goulden, Holden, Jendralski, Dolffus, Strumpell, Dreschfeld, Raverdino, von Gehuchten, Bouchut, Dechanne and Bogaert. A good recent summary of the subject is that of Salvati.

COURSE AND SYMPTOMATOLOGY

Clinically, the disease is one of bilateral optic neuritis, acute or subacute, followed in a variable length of time by myelitis. Occasionally the ophthalmoscope does not help directly, because, as was true in one of my cases, only the signs of retrobulbar neuritis are present. Ocular movements and pressure on the bulb may elicit severe pain, but this phenomenon is also sometimes observed in retrobulbar neuritis due to other causes. The neuritis may be followed by complete blindness or only impairment of vision with restriction of the visual field for white and for colors and occasionally with central scotomas. Return of vision is always slow, but occurs in about from 50 to 60 per cent of cases. A marked discoloration of the papilla persists in spite of the improvement. In certain cases paralyses of all the muscles of the eyeball have occurred (case of Henneberg-Karschensteiner, Makian and Bielschowsky). The acute form is most frequent. Vision is rapidly impaired within a few days to twenty-four hours, often to complete suppression of perception of light. The patient may complain of very severe pain in the orbit, which is made worse by ocular movements or digital pressure. There are usually no external signs. The ophthalmoscope reveals a more or less intense optic neuritis sometimes with

From the Department of Ophthalmology, Cook County Hospital.

small hemorrhages on the papillary border and light turbidity in the macular region. The two eyes may be involved simultaneously, or one eye may be affected a few days or weeks after the other (in the cases of Steffan and Dreschfeld, one and two months afterward, respectively). Blindness is usually complete and remains so for a variable period of time, from a few days to a few months. First, perception of light returns, and gradually vision improves until it finally becomes normal or nearly normal, unless the patient succumbs to the coexisting myelitis. The mortality is 50 per cent. It is important to note that the myelitis may be entirely independent of the ocular condition; thus, the optic neuritis may become better while symptoms of myelitic involvement progress. In nearly three fourths of the cases neuritis preceded the myelitis; in other cases neuritis and myelitis occurred simultaneously, while in a few exceptional cases the myelitis preceded the neuritis (Seguin, Katz, Noyes and Gallovardini). Of note are the observations of Stephan Erb, who reported a recurrence of this condition twice in the same person, and of Dreschfeld, who reported a recurrence in one eye. In these two cases the myelitis had not yet resolved at the time of the recurrence. The interval between the onset of ocular symptoms and the myelitis varies from twelve hours, as in the case of Chisolm, to eighty-one days, as was reported in the case of Steffan. The visual fields present a variety of defects: temporal hemianopia, central scotoma or central scotoma combined with concentric narrowing. The central scotoma may be absolute or relative. The color fields show a defect for green and red.

ETIOLOGY AND PATHOGENESIS

The etiology of optic neuromyelitis is still obscure. In the cases of Dreschfeld and Alexander the cause seemed to be syphilis. In the fifteen cases collected by Katz, syphilis was considered a predominating factor in five of the cases, and nephritis in one of the cases. In the case of Achard and Guineau, it was alcoholism. Tuberculosis and neurodiathesis have also been mentioned. The majority of cases occur in persons in perfect health. In a case of Gessner's the symptoms of myelitis were overshadowed by blindness following a hemorrhage. Bacteriologic researches up to the time this paper was written have amounted to naught. No plausible explanation has as yet been advanced to account for the fact that the optic nerve and central gray matter only are attacked while the remaining cranial nerves are spared. However, the hypothesis has been advanced that there might be a vasomotor action exerted by a disease of the gray matter of the optic nerve and by a destruction of the trophic centers of these nerves by the progressive myelitis. This would tend to substantiate the fact that in the majority of cases the neuritis precedes the myelitis. In nearly fifteen autopsies

a similar alteration in the nerve and eye was found without any direct continuity between the two focal processes. However, Weyl and Gallarvardin mentioned diffuse encephalitis, implying a continuous pathway between the two foci. The changes in the optic pathway affect the chiasm, the nerve sheaths and the optic nerves up to the retrobulbar portion. Microscopically, there is a leukocytic infiltration of all the layers of the optic nerve, its connective tissue and even its blood vessels. The optic tracts are degenerated and show fatty infiltration, being replaced by neuroglia tissue. In the more serious cases these changes affect all parts of the nerve and in others, only the central portion, while in another group only an isolated portion of the nerve may be involved. This accounts for the variation in the visual fields. Salvati considered the process as that of an interstitial inflammation of the parts first affected, with replacement by a regeneration of neuroglia cells. In the retina the central degeneration is manifested by an extreme thinning of the nerve fiber layer and a diminution of the ganglion cells (Elschnig, Dalen).

PROGNOSIS AND THERAPY

In 50 per cent of the reported cases the patients recovered, and in all of these the vision returned to normal or nearly normal.

The therapy is not certain, as the etiology is unknown, and has consisted in the use of mercurial preparations, iodides, salicylates, strychnine, foreign protein therapy, electrical therapy and shaded lenses for the photophobia, if present.

REPORT OF CASES

On account of the rarity of the condition, and especially because of the paucity of reports concerning it in the English literature, the following case seen at the Cook County Hospital is reported and notes on a second case are given.

CASE 1.—*History.*—A girl, white, aged 5 years, was first seen in the outpatient department of the ophthalmologic division where she was brought by her mother because of poor vision and lameness of one leg. Examination at this time revealed bilateral papillitis and no perception or projection of light in either eye. She was, therefore, admitted to the children's department of the Cook County Hospital for further study and observation on April 11, 1932.

Except for the fact that she had contracted measles three years previously, the child had always been healthy until one month before admission, when she had a bad cold. At this time she complained of a headache, aching of both ears but no discharge, a sore throat and fever, ranging around 102 F. for two or three days. Emesis was not present. The child was kept in bed for one week. When she was allowed to be up she complained of aching legs and walked "stiff-legged." After this attack, which lasted about two weeks, she seemed normal. She appeared thin and emaciated, since she had lost about 10 pounds (4.5 Kg.) in the interval.

On April 9, the child complained of a frontal headache. On April 10, after her usual afternoon nap, she asked her mother if the light was turned on. The mother then realized that the child could not perceive light. This was the first intimation of any visual disturbance. On April 11, the child seemed weak on the right side and dragged the right foot in walking. Inventory by systems revealed no pertinent findings not already stated. The past history was not significant, and the family history was unimportant. Both mother and father denied having had a venereal disease. A careful physical examination gave negative results except for the neurologic and ophthalmologic findings.

Neurologic Examination.—This child was remarkably cooperative, and consequently the findings are of more value than those in the average child of 5.

Motor System: The patient could not walk without some help. The gait was unsteady and on a wide base, and the right foot was dragged a little, with a slight foot drop, making a steppage gait. Coordination was normal, all the tests being done fairly well. Skilled acts were done well with either hand. There was no adiadokokinesis. Muscular strength was markedly diminished for all movements of the right leg. In the upper extremities it was equal. Flaccidity of the right lower extremity was present.

There was no rigidity or atrophy. The Brudzinski sign was positive on both sides. All the other signs were negative. All the deep reflexes were slightly exaggerated, and the superficial ones were all normal. No pathologic reflexes were elicited.

Sensory System: Touch, pain, temperature sense and stereognosis were normal. All the cranial nerves were normal with the exception of the optic and abducens nerves.

Laboratory Findings.—Examination of the spinal fluid showed the following: pressure, normal; Queckenstedt test, normal; color, clear; Pandy reaction, positive; cell count, 92 per cubic centimeter; smear, predominance of lymphocytes; Kahn test, negative; culture, no growth, and sugar, 50 mg. per hundred cubic centimeters.

The Kahn reaction of the mother's blood was negative.

The blood count was 13,800 white blood cells, with 68 per cent polymorphonuclears, 2 per cent eosinophils, and 30 per cent lymphocytes.

The Pirquet test was negative.

The urine was normal.

The ocular examination showed a slight convergent strabismus due to a slight paresis of the external recti. The pupils were equal and regular and reacted to light and in accommodation. The remainder of the external eye was normal.

Examination of the fundus showed the borders of the right disk to be clearly defined; the veins were tortuous. There were no exudates or hemorrhages. The borders of the left disk were elevated and very indistinct. The veins showed a moderate degree of tortuosity. These findings suggested retrobulbar neuritis on the right side and optic neuritis on the left.

Treatment consisted of inunctions of mercury, 30 per cent daily, and the use of potassium iodide.

Progress.—The child's vision gradually improved so that by the time she left the hospital she could perceive and project light accurately. She was temporarily lost from observation after she was dismissed from the hospital, but was finally located in Flat River, Mo. A letter from Dr. C. H. Appleberry, dated Feb. 8, 1933, nine months after the illness, yielded the following information:

"Miss H. at the present time is doing very nicely, is attending school regularly and has gained 6 pounds in weight. Physical examination showed the pupils to be round and regular; they react to light, in accommodation and consensually.

"Ophthalmoscopically, the retinal vessels are normal in size. No choked disk is present. The vision in the left eye is 20/20; in the right, 20/40. The throat and chest are normal. The abdominal reflexes are equal and active. The extremities are normal. The reflexes are normal, and no pathologic signs are present in the toe."

In a personal communication Dr. Sanford R. Gifford cited the following case:

CASE 2.—A boy, aged 12, on presenting himself gave a history of rapid loss of vision in the left eye for the past few days. Vision was 20/20 in the right eye; there was no perception of light in the left eye. The right fundus was normal. The left showed a marked swelling of the disk with tortuosity of the veins and peripapillary edema. There were no symptoms of neurologic disturbance, and the boy was in apparently good health. As a possible cause of this unilateral papillitis a tumor of the orbit or of the nerve itself was considered, although no exophthalmos was present. Roentgenograms of the sella turcica and of the optic foramina showed a normal condition. A week later, while walking, the boy fell and was unable to walk; when seen at the hospital he had a right-sided paralysis. Dr. G. A. Young pronounced the condition acute myelitis. Vision in the right eye now became much impaired, though ophthalmoscopic findings were negative. Under foreign protein therapy, vision began to return in the right eye, and then in the left eye, while the paralysis also cleared up. When the child was examined at Dr. Gifford's office two months later, vision was 20/20 in the right eye and 20/20 in the left. The right disk was slightly pale; the left showed postneuritic changes and more marked pallor than the right. The left field showed only moderate concentric contraction.

The case of Roger, Opiu and Sédan is mentioned because the first symptom was a paralytic disturbance of both lower extremities. Ophthalmoscopic findings were always normal, but the vision of both eyes was greatly impaired. The authors reported the case because of the unusual precedence of the symptoms of the spinal cord and their relative unimportance. In another case reported by Marinesco, Draganesco, Sager and Grinesco, sections showed differences from multiple sclerosis but also some resemblances. Milian and his co-workers reported the case of a man who gave negative reactions to tests of the blood and spinal fluid and during the following days proved to have signs of paralysis in the lower limbs and visual disturbances. The visual difficulties consisted in a decrease of visual acuity with edematous papillitis accompanied by a central scotoma for colors. In spite of treatment, the paralysis increased; the spastic manifestations and accentuation of the sensitivity were the two chief manifestations. Appearance of an ascending urinary infection was concomitant with sphincteral dystrophies complicated by pulmonary and

renal symptoms, which led to death in from six to seven weeks after the onset. The authors do not agree with other workers that this disease is a syndrome.

Dedimos reported two cases of acute optic neuromyelitis associated with malaria. On quinine therapy the vision of the second patient improved from 0 in both eyes to 0.2 in the right eye and 0.4 in the left. In both cases the ocular symptoms came on first, followed by symptoms referable to the spinal cord. The first patient died with no vision in either eye.

I have noted that the symptom complex described by the European writers as optic neuromyelitis is in some respects similar to multiple sclerosis with its associated retrobulbar neuritis. The latter has a chronic course, and while the visual defects may come on acutely and subside rapidly, the disease is prone to recur and leave permanent damage. In optic neuromyelitis, the course is very acute, ending either in death or in practically complete recovery from both general and visual changes.

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VELONOSCOPY CHECK TEST FOR ASTIGMATISM

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Velonoskiascopy or, more briefly, velonoscopy, is a subjective method of examination which in a sense is a complete method. By this I mean that it can be used to determine and measure all errors of refraction and accommodation, including hyperopia, myopia, astigmatism and presbyopia. But like most other general tests it is more effective and more reliable in some of its applications than in others. I wish to call attention to only one of its applications, namely, its use as a check test for astigmatism. The method was designed by Professor Lindner of Vienna about eight years ago. I have modified the test somewhat in order to make its application more workable in routine practice.

The place of the test in an examination will be more readily understood from an illustration. Suppose that one has obtained by the usual methods a correction in the trial frame which is +2.00 sphere ⊃ +1.00 cylinder, axis 90. In any spherocylinder combination it is possible that there may be a slight malcorrection of either the sphere or the cylinder or, more likely, of both, especially in the relative distribution of the sphere and cylinder. That is, the finding of a +2.00 sphere ⊃ a +1.00 cylinder may be an approximate finding for what should be either a +1.75 sphere ⊃ a +1.25 cylinder or a +2.25 sphere with a +0.75 cylinder. That is, there was an overcorrection of one element at the expense of the other. The velonoscopy test offers a means of checking the accuracy of either the sphere or the cylinder or of both, but primarily of the cylinder.

The apparatus used consists of a few charts and a few wire crosses. The main chart is one having a large revolving white cross on a red background, with a surrounding dial marked off in 5 degree intervals. There are two small auxiliary charts like this. The wire crosses consist merely of two thin black wires at right angles, mounted in a rim and either placed on a handle to be held by the patient or set in a ring to fit a trial frame. I have three such crosses, the thickness of the wires being 1, 1.5 and 2 mm., respectively. For the average pupil of from 4 to 5 mm. I use the 1.5 mm. cross. For small pupils the 1 mm. cross, and for large pupils the 2 mm. cross, is preferable.

The method of procedure is most easily explained by a few illustrations. Suppose that there is in the trial frame, as has been previously

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mentioned, a +2.00 sphere ⊃ a +1.00 cylinder, axis 90, the accuracy of which is to be checked. The eye is fogged about 1 diopter; that is, the +2.00 sphere is replaced with a +3.00 sphere. The chart is set up on a level with the patient's eyes, about 20 feet away, with the cross-lines at 90 and 180 degrees. The wire cross is set in the trial frame in the cell farthest from the patient's eye, also at 90 and 180 degrees. As the patient looks through the wire cross at the cross-lines on the chart he will see a red streak passing through each white line; that is, he will see a vertical and a horizontal red streak passing through the vertical and horizontal white lines. If the trial frame is not accurately centered he may have to make some slight adjustments of the head in order to see the two red streaks at the same time. Incidentally, if he has to make

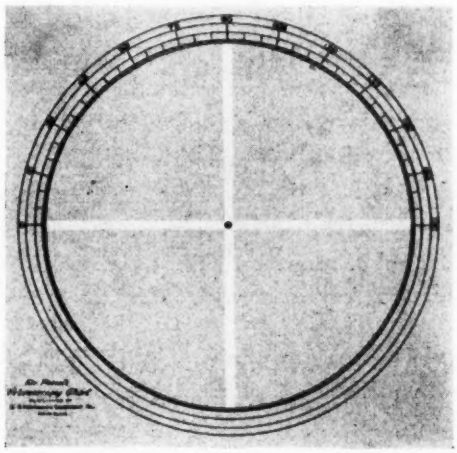

Chart with revolving white cross, used in velonoscopy check test.

excessive adjustments of the head in order to see both streaks at the same time it will call attention to the fact that the trial frame is poorly centered and that previous tests may need revision.

When the patient sees the two red streaks he is asked whether they are equally wide or whether one streak is wider than the other. If the streaks appear equally wide, the cylinder is approximately correct. I say "approximately" because the check up is continued. The fog is reduced by changing the +3.00 sphere to a +2.75 sphere; again the patient is asked to compare the width of the two streaks. Both streaks will now appear narrower, and a slight difference may be more easily noticed. If the streaks again appear equally wide, the fog is reduced still further until they disappear. If the two streaks appeared equally wide all through the reduction of the fog but if, in order to eliminate the streaks, the fog had to be reduced by 1.25, the cylinder was correct but the sphere was a little too strong. The correct prescription should be

+1.75 ⊃ +1.00, axis 90. If, on the other hand, during some stage in the reduction of the fog the patient said that the two streaks were not equally wide, the 1 diopter cylinder is incorrect, irrespective of the accuracy or inaccuracy of the sphere. The cylinder is corrected by bringing about equalization of the two red streaks. This can be obtained either by widening the narrow streak or by reducing the wider streak.

As a general rule, in order to widen a streak a plus cylinder with its axis corresponding to the streak is used; i. e., to widen a vertical streak one must use a plus cylinder, axis 90, and to widen a horizontal streak, a plus cylinder, axis 180. To reduce the width of a streak one must use a minus cylinder, axis likewise in the direction of the streak. That is, to reduce the width of a vertical streak one must use a minus cylinder, axis 90, and to reduce the width of a horizontal streak, a minus cylinder, axis 180.

Professor Lindner's method is to use a minus cylinder in order to reduce the wider streak. The method which I use is to change the streak which corresponds to the axis of the cylinder already found, whether this is plus or minus. I call this the axis streak. If the axis is 90, the vertical streak is the axis streak. If the axis is, say, 30 degrees, the 30 degree streak is the axis streak, and so on. In other words, attention is paid primarily to the axis streak, which is widened with a plus cylinder or reduced with a minus cylinder, as the case requires.

As an illustration of the method, the original example may be considered, in which the correction was approximately +2 ⊃ +1.00, axis 90. As the axis of the trial cylinder is 90 degrees, it is noted that the axis line or the axis streak is at 90, and special attention is paid to the vertical streak. Suppose that, after the eye has been fogged with a +1.00, with the cross-lines and wire cross properly placed, the patient says that the horizontal streak is wider. This means that the axis streak, i. e., the vertical streak, is narrower. A plus cylinder is needed to widen it, so the +1.00 cylinder, axis 90 is replaced with a +1.25 cylinder, axis 90, and the patient is asked to note the effect. If the streaks appear equally wide the fog is reduced while the patient watches the two streaks until they entirely disappear. Suppose that, as the streaks become narrower, he again notes that the horizontal streak is a little wider. The +1.25, axis 90 is replaced with a +1.50, axis 90, and the effect is noted. If, as a result of this change, the vertical streak appears wider, the cylinder lies between +1.25 and +1.50, and those who apply the scale of an eighth of a diopter can use a +1.37, axis 90. The examination is completed by reducing the fog still further until the two streaks disappear.

As a further illustration, suppose that, with the setup just described, the patient said that the vertical streak, which is the axis streak, was wider. Minus cylinders are needed to reduce it. So the +1.00 cylinder, axis 90 is replaced with a +0.75 cylinder, axis 90. If this change causes a reversal, in the sense that the vertical streak is now narrower, the cylinder required is one between a +1.00 and a +0.75, axis 90, the nearest to which is a +0.87, axis 90. I may add that instead of immediately replacing a cylinder with another stronger or weaker one, it is often advisable to first hold a + or —0.25 diopter cylinder over the other cylinder and note the effect.

In the two illustrations cited one will note that the + cylinder was increased in order to widen the axis streak or decreased in order to reduce it until the two streaks appeared alike. This procedure was continued while the fog was reduced to a point at which the streaks entirely disappeared. Since attention is paid primarily to the axis streak, the physician can, if he chooses, confine his questions to the patient primarily to those regarding the axis streak; that is, he can ask the patient at the beginning whether the vertical streak is narrower or wider than the horizontal streak and then proceed directly to change the vertical streak by adding a plus cylinder if it is narrower or a minus cylinder if it is wider.

I have used two illustrations involving a plus cylinder, but the same procedure is carried out with a minus cylinder in the trial frame or phoroptor. Suppose that there is a +2.00 ⊃ —1.00, axis 180. The eye is fogged by substituting a +3 for the +2.00; the cross-lines and wire cross are properly placed, and mental note is made that the axis streak is the horizontal 180 degree streak. If the horizontal streak appears wider, a minus cylinder is needed, and the —1.00, axis 180 is changed to a —1.25, axis 180. If the horizontal streak is narrower, a plus cylinder is needed; the —1.00 cylinder is changed for a —0.75 cylinder, axis 180, and the procedure is continued as outlined before.

In my illustrations I used the 90 and 180 positions because they are most common. But the test works equally well for every other position. Suppose that the trial frame contains a —1.00 sphere ⊃ a —1.00 cylinder, axis 70. The cross-lines of the chart are turned to 70 and 160 degrees, and the wire cross is placed at 70 and 160. The eye is fogged by removing the —1.00 sphere, and mental note is made that the axis streak is the 70 degree streak. The patient is asked to compare the two streaks as before; if they appear unlike, the width of the 70 degree streak is changed by increasing or decreasing the minus cylinder, axis 70 until the two streaks are equally wide and remain so to the point of their disappearance as the fog is gradually removed. Incidentally, this method may be used, if desired, to check the accuracy of the glasses worn by a new patient who comes for examination. A +1.00 sphere

is placed over the old lenses; the cross-lines and wire cross are set up as previously described, and by comparison of the two streaks the accuracy or inaccuracy of the patient's glasses may be roughly determined.

In conclusion, I may say that the velonoscopy check test has the advantage of enabling the patient to make a simultaneous comparison of the things he is asked to compare. The two streaks which he compares are both before his eye at the same time. He is not asked to compare one impression with the memory of another impression, as in most tests. Furthermore, the test is equally applicable to the literate and illiterate, to the intelligent and the less intelligent. For the latter and, in general, to facilitate work and save time, the physician may use the auxiliary charts in order to acquaint the patient with the purpose of the test. He tells the patient that he will see two red streaks either equally wide, as on the one chart, or one streak wider than the other, as on the other chart. Finally, I would say that, next to the cross-cylinder test which is the king of all check tests, the velonoscopy test is simple and effective and is a worth-while addition to the equipment of any physician who is interested in fine refraction.

500 West End Avenue.

Ophthalmologic Review

COLOBOMA OF THE OPTIC NERVE ASSOCIATED WITH POSTERIOR LENTICONUS

JAMES S. SHIPMAN, M.D.
CAMDEN, N. J.

Coloboma of the optic nerve is a rare, congenital anomaly, especially when it is unassociated with defects of the surrounding choroid. Vossius [1] found this condition present in only three of twelve thousand persons examined. Caspar,[2] in 1887, collected reports of twenty cases, and Saemisch and his co-workers,[3] in 1891, increased this number to forty-eight. Coats,[4] in 1907, added six more cases, in all of which he had made microscopic studies and verified the diagnosis.

In 1912, Crampton,[5] in an extensive review of the literature, could find only twenty-one cases of true coloboma of the optic nerve, and these occurred in sixteen patients. Included in this group were the six unequivocal cases of Coats,[4] together with three cases of his own. Since that time other cases have been reported. Calhoun,[6] in 1930, reported a bilateral case associated with holes in the disk and a cyst of the sheath, of the optic nerve, in which microscopic studies were made. He stated that he could find only twenty-six cases in addition to the twenty-one found by Crampton, and in only one of these twenty-six were microscopic studies made, i. e., one reported by Rochat [7] in 1913. Among the other men in this country who have reported cases of this anomaly are Chance,[8] Jennings,[9] Lerner [10] and Schlindwein.[11]

1. Vossius, A.: Beitrag zur Lehre von den angeborenen Conis, Klin. Monatsbl. f. Augenh. **23**:137,1885.

2. Caspar, J. H. R. L.: Ueber das Colobom des Sehnerven, Bonn, J. Bach, 1887.

3. Saemisch; Caspar, and Krüger, in Festschr. zur Feier von des siebzigsten geburtstages von Hermann von Helmholtz, Leipzig, L. Voss, 1891, p. 1.

4. Coats, G.: The Pathology of Coloboma of the Nerve Entrance, Roy. London Ophth. Hosp. Rep. **17**:178, 1907.

5. Crampton, G. S.: Two Cases of Binocular Coloboma of the Optic Nerve in the Same Family, Tr. Am. Ophth. Soc. **13**:451, 1913.

6. Calhoun, F. P.: Bilateral Coloboma of the Optic Nerve, Arch. Ophth. **3**:71 (Jan.) 1930.

7. Rochat, G. F., quoted by Calhoun.[6]

8. Chance, B.: A Case of Coloboma of the Optic Nerve, Tr. Coll. Phys. Philadelphia **36**:317, 1914.

9. Jennings, J. E.: Coloboma of the Optic Nerve, Am. J. Ophth. **7**:788 (Oct.) 1924.

10. Lerner, M. L.: Coloboma of the Optic Nerve, Am. J. Ophth. **9**:241 (April) 1926.

11. Schlindwein, G. W.: Coloboma of the Right Optic Disk in a Twelve Year Old Child, Pennsylvania M. J. **33**:162 (Dec.) 1929.

Certainly some of the cases reported as true coloboma of the optic nerve have been associated with defects in the choroid, lens and iris, and it is possible that some have been exaggerated glaucomatous cups, and were therefore not included by Crampton [5] and Calhoun [6] in their forty-eight cases.

Crampton suggested a convenient classification of cases into two groups: those in which ophthalmoscopic examination shows no choroidal fault in the margin of the pseudodisk and those cases in which microscopic examination indicates the true nature of the defect.

PATHOGENESIS OF COLOBOMA OF THE OPTIC NERVE

Coloboma of the optic nerve is accounted for by different theories, the first and most accepted of which is: some interference with the normal closure of the fetal cleft, first propounded by von Ammon [12] in 1831, and confirmed by von Hippel's [13] experiments. Intra-uterine inflammation as the cause was suggested by Deutschmann [14] in 1881, and Fournier [15] believed that toxic effects of tuberculosis, alcohol and syphilis might be responsible, but neither of these two theories have been considered very strongly.

Collins and Mayou [16] described the fetal cleft theory clearly:

> The fetal ocular cleft normally closes in early fetal life and cuts off the mesoblastic structures external to the secondary optic vesicle from those internal to it. In some animals this does not become closed, and we have a permanent union of the mesoblastic structures inside and outside the eyeball, as evidenced by the falciform process in fish, and the pecten in birds. This same union of the mesoblast external to the secondary optic vesicle, with that internal to it, occasionally occurs in man in the region of the ocular cleft, and prevents the edges of the secondary optic vesicle from coming together, and causes a coloboma of the retina. In these eyes there is an abnormal development of fibrous tissue in the vitreous chamber, by extension thru the cleft, and microphthalmus results.
>
> A delay in separation of the mesoderm outside from that inside the eye, without permanent union, may also result in failure in the closure of the cleft in the secondary optic vesicle, and the formation of a coloboma. When this occurs the changes are not limited to the structures derived from the secondary optic vesicle, but involve also those immediately external to it. If the defective closure is in the extreme posterior part of the cleft, there is a coloboma of the optic nerve sheath; if somewhat further forward, a coloboma of the choroid; or if near the anterior lip of the cup, there is a coloboma of the ciliary body and the iris which normally grow from it.

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12. von Ammon, F. A., cited by Lerner [10] and Parsons.[17]

13. von Hippel, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1900, vol. 2, p. 27.

14. Deutschmann, cited from Parsons.[17]

15. Fournier, cited from Lerner.[10]

16. Collins, E. T., and Mayou, M. S.: Pathology and Bacteriology of the Eye, Philadelphia, P. Blakiston's Son & Co., 1911, pp. 20, 26 and 27.

Parsons [17] stated that the shape of a coloboma of the optic nerve is usually round or vertically oval. The size is from two to twenty times that of the normal disk. Total excavation is uncommon, and the lower part of the disk is usually deeper.

Collins and Mayou [16] stated that many colobomas of the choroid reach up to the nerve, and due to ectasia the lower border of the nerve is displaced upward and backward, the surface of the nerve facing downward; in such cases the sheath of the nerve is not involved in the coloboma.

Van Duyse [18] gave the following characteristics of colobomas of the optic nerve: (*a*) enlargement of the papillary area, with irregularity of the lower margin; (*b*) partial or total excavation, the greatest depth being in the inferior portion; (*c*) glistening white surface, alternating with shades of gray in the excavated portions, and (*d*) special disposition of vessels.

Casper [2] classified the arrangement of the vessels definitely into three classes: (*a*) cases in which all vessels emerge from the lower part of the pseudodisk, even those which subsequently turn upward; (*b*) cases in which the vessels emerge at or a little above the center and are nearly normal in arrangement, and (*c*) cases in which the vessels appear at the edges around the whole circumference of the disk. The vessels in the last group are usually referred to as cilioretinal vessels.

Beard [19] stated that often the excavated portion is surrounded by what seems to be an exaggeration of the pigment and scleral rings, and when the whole area within the scleral ring is depressed, he likens the condition to that of a magnified glaucomatous excavation.

Collins and Mayou [16] stated that in coloboma of the nerve alone the nerve entrance is practically of normal dimensions, and all abnormalities are within its boundaries. A fold of retina is found passing through the lamina cribrosa and hollowing out the nerve. Pathologically coloboma of the nerve can be differentiated from one involving both the choroid and the nerve by the relation which the excavation bears to the intervaginal space in the sheath of the nerve. If the defect is on the inner surface, it is a coloboma of the nerve, and if it is on the outer surface, it is a coloboma of the choroid. This point of differentiation, as well as the position of the retinal vessels as they emerge from the pseudodisk, was stressed by Coats.[4]

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17. Parsons, J. H.: Pathology of the Eye, London, J. & A. Churchill, 1907, vol. 3, p. 830.

18. Van Duyse, cited from Beard.[19]

19. Beard, in Pyle, W. L.: An International System of Ophthalmic Practice, Philadelphia, P. Blakiston's Son & Co., 1913.

Manz,[20] in 1891, made microscopic sections of a human eye in which the diagnosis of coloboma of the optic nerve had been made ophthalmoscopically, and his examinations proved what the ophthalmoscope had made seem probable; i. e., that the central vessels which appear behind the margins of the coloboma do not pass to the retina from the optic nerve, but from outside the nerve.

The vision reported in the cases of coloboma of the optic nerve varies. In some it is normal, and in others it is only perception of light. In the bilateral cases, and according to Beard [19] these are not so uncommon, it is usual to find one eye almost blind and the other nearly normal. This was true in Crampton's [5] cases, although both eyes in Chance's [8] case showed 5/15 vision after the inflammatory symptoms had cleared up.

Crampton's [5] cases are unusual in that they were bilateral and occurred in a brother and sister, the right eye in each being almost blind and the left eye having almost normal vision. Certainly the coloboma in the left eye of his first patient, and in each eye of his second patient, appear to be a true coloboma of the optic nerve.

Neiden,[21] in 1879, was apparently the first to describe a case of true coloboma of the optic nerve or its sheath. He described three cases, although he did not include the histologic changes.

In 1897, Goerlitz [22] published what seems to have been the first histologic study of a coloboma of the optic nerve uncomplicated by any defects in the choroid, although Coats [4] had some doubt about this being a case of true coloboma of the optic nerve.

In 1898, von Hippel [23] reported what he believed to be a case of true coloboma of the optic nerve. Eight years later (1906), Parsons and Coats [24] published the report of a case of the same type, and Coats [4] included this in his article with a full description of his other cases.

Many cases of coloboma of the optic nerve have been reported associated with coloboma of the choroid, lens or iris, and some have been reported with other anomalies—persistent hyaloid, by Becker,[25]

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20. Manz, W.: Ueber das angeborene Colobom des Sehnerven, Arch. f. Augenh. **22**:1, 1891; On Congenital Coloboma of the Optic Nerve, Arch. Ophth. **21**:263, 1892.

21. Neiden, A.: Coloboma of the Sheath of the Optic Nerve, Arch. f. Ophth. **8**:501, 1879.

22. Goerlitz, M.: Anatomische Untersuchung eines sogenannten Colomba nervi optici, Arch. f. Augenh. **35**:219, 1897.

23. von Hippel: Arch. f. Ophth. **35**:219, 1897; **45**:317, 1898.

24. Parsons, J. H., and Coats, G.: Brain **29**:209, 1906.

25. Becker, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1877, vol. 5.

Van Duyse [26] and others; remnants of the posterior portion of the sheath of the lens, by Pfanmüller,[27] and opaque nerve fibers, by Würdemann.[28] I can find only one case reported in which a posterior lenticonus has been associated with a coloboma of the optic nerve. This was reported by Van Duyse, and was in an infant, 5 days of age, with a lateral proboscis and a typical coloboma of the lid, iris, choroid and nerve. Microscopic sections were made, which showed a true posterior lenticonus. Van Duyse [29] found a concentric contraction of the visual field in cases of coloboma of the optic nerve, and Neiden [30] found a contraction of the upper field. Jennings found a centrocecal scotoma in his case.

REPORT OF A CASE

History.—A well developed Polish boy, 13 years of age, was referred to the ophthalmic clinic of the Cooper Hospital by the school authorities for examination, and was first examined by me on June 6, 1931. The family and previous medical histories were essentially unimportant. The vision in the right eye, uncorrected, was 1/60, and was not improved by refraction. The vision in the left eye, uncorrected, was 6/6, and was improved to 6/5 with +0.75 sph. ⊃ —0.25 cyl., axis 75. The eyes were of normal size and showed no squint or nystagmus. Each cornea measured 13 mm. The pupils were round, regular and equal, and reacted normally to light. The irides were light blue and healthy in appearance. The tension was normal to palpation in each eye.

Ophthalmoscopic Examination.—The right eye, when viewed with a + 8 D. lens, gave the appearance of an oil globule in the posterior portion of the lens so characteristic of posterior lenticonus, its movement being definitely against the rule when the patient moved the eye up and down. This refractive globule was about 4 mm. in diameter, and was definitely and sharply demarcated at its margins, although a few small opacities could be seen within the limits of the globule. The vitreous showed a few stringy opacities. The fundus was easily seen through the periphery of the lens with a —1 D. lens, but when viewed through the center it was necessary to use a —15 D. lens to bring the fundus into clear focus. The most striking part of the fundus was the pseudo-optic disk, which appeared to be about twice the normal size and markedly excavated from 5 to 10 D. It was grayish white, and the margins were sharply defined, the temporal side showing a thin ring of choroidal pigment. The vessels from below bent sharply at the margin and were lost to view; those from above gradually sloped in and passed across the upper floor of the excavation to the central portion, where they were lost. The vascular distribution from the margins out to the periphery was normal, and no gross lesions were noted in the periphery or the macula (figs. 1 and 2). In the left eye the media were entirely clear, and the fundus was healthy in all respects.

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26. Van Duyse, quoted from Parsons.[17]

27. Pfanmüller, F.: Dissert., Bonn, 1894.

28. Würdemann, H. V.: Centralbl. f. prakt. Augenh. **20**:533, 1896.

29. Van Duyse, G. M.: Proboscide latérale et colobome oculaire atypique avec lenticone postérieur, Arch. d'opht. **36**:463, 1919.

30. Neiden, quoted from Parsons.[17]

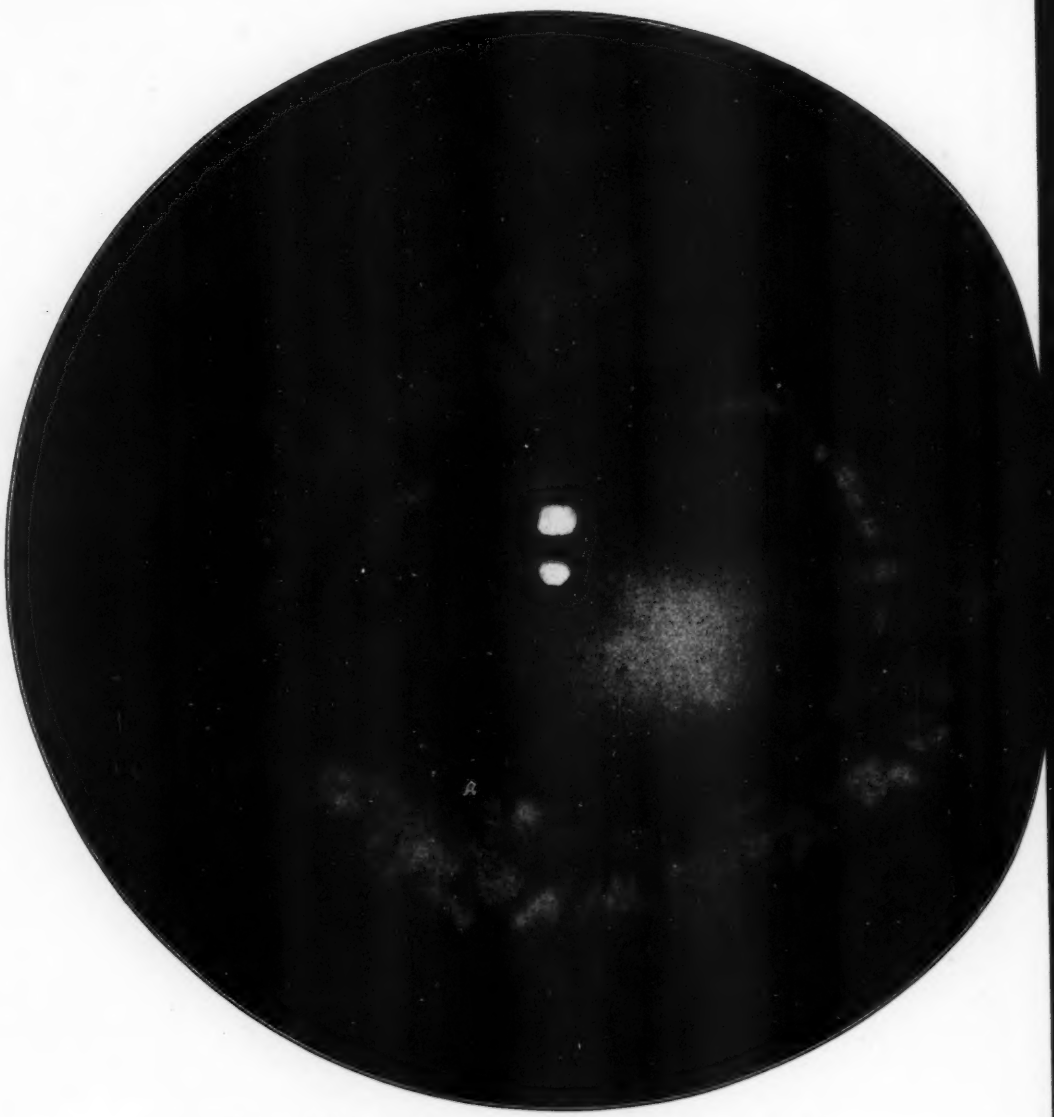

Fig. 1.—Enlarged photograph of coloboma of the optic nerve.

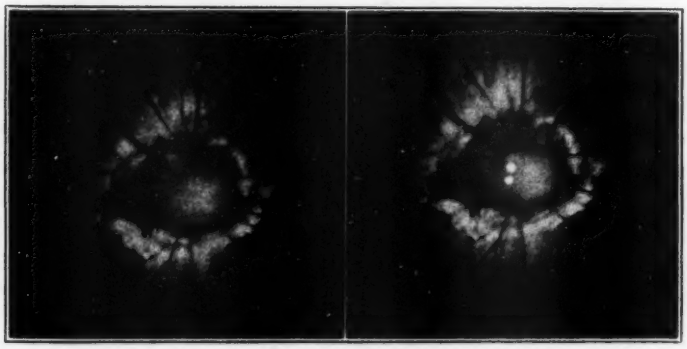

Fig. 2.—Stereoscopic photographs of coloboma of the optic nerve.

The visual fields showed a large centrocecal scotoma and a moderately marked contraction of the form field (figs. 3 and 4).

Study of the right eye with the slit lamp, as reported by Dr. Cowan, revealed the following: The cornea was clear. The anterior portions of the lens showed no

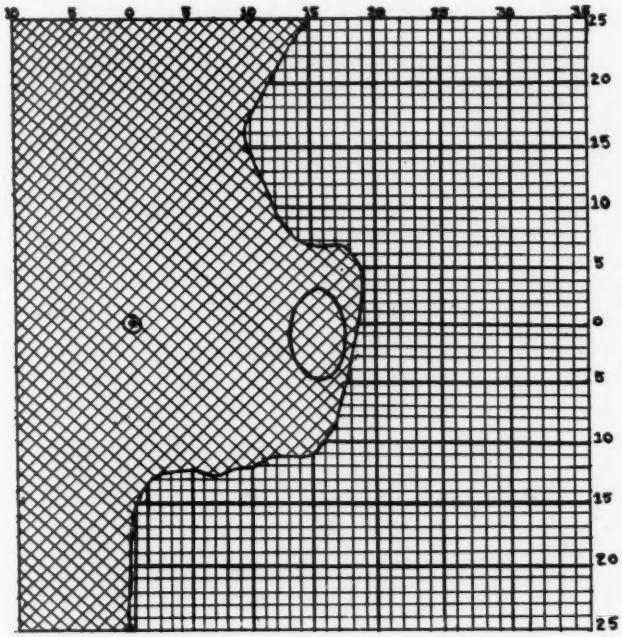

Fig. 3.—Central field in a case of coloboma of the optic nerve of the right eye. There was central scotoma; test object, 1½°.

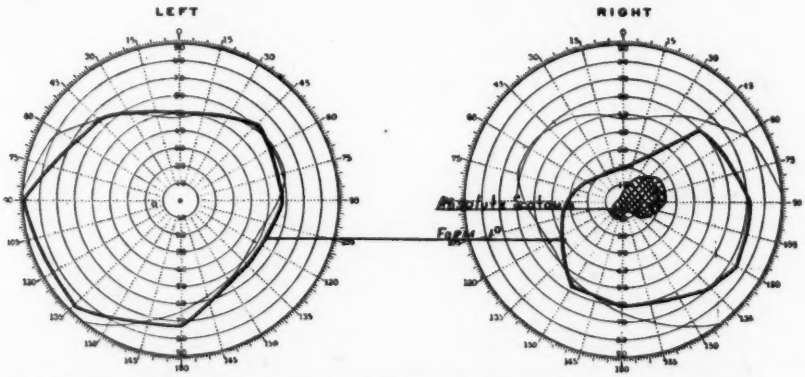

Fig. 4.—Form fields in a case of coloboma of the optic nerve of the right eye.

abnormality. The anterior and posterior Y-sutures were intact. There was a slight backward bulging of the posterior face of the embryonic nucleus, with an increase in relucency, and from here backward there were several optically empty areas forming an irregular line toward the posterior capsule. Just inside the posterior capsule there was a dense, white, spindle-like opacity extending horizontally. The middle of the posterior portion of the lens bulged backward.

The bulge was at the pole, but was inclined somewhat to the nasal side. The double capsule lines were not disturbed (were parallel), unless they were a trifle wider apart than normal. The bulging was aspherical, making a gradual curve neither of the true type of Globus nor that of Conus. Corresponding to what would be the base of the protrusion, there was a thin, sharp, white line forming a large circle on the posterior surface of the lens. At the posterior pole on the surface of the lens there was a thin, yellowish, glistening area which gave a play of colors, and in which there was an area that seemed to be optically empty. Extending away into the vitreous, past visibility, was a cometlike, grayish membrane with its apex attached to the vertex of the protuberance on the lens. There were numerous opacities in the vitreous.

POSTERIOR LENTICONUS

According to the literature, lenticonus is a rare condition, although the posterior type is more common than the anterior type. Tyson,[31] in 1928, reduced the number of true cases reported to six. Reese,[32] in the same year, reported two true cases, and Butler,[33] in 1930, reported six more cases.

The typical ophthalmoscopic picture of true posterior lenticonus has been well described by Reese,[32] in an excellent review of the literature on this subject, as:

a round refractive globule, two to three millimeters in diameter, simulating a drop of oil, at or near the posterior pole of the lens. Its margins are quite sharply circumscribed, and it moves against when the eye is moved. The slit lamp gives the definite diagnosis when posterior protrusion of the lens surface is noted. True posterior lenticonus is rarely bilateral, while false posterior lenticonus is usually bilateral. Nuclear sclerosis in the aged accounts for many false types of posterior lenticonus. Posterior lentiglobus is somewhat similar to posterior lenticonus, tho its protrusion is more definitely and abruptly demarcated from the rest of the lens.

Theories as to the causes of posterior lenticonus are: hyaloid traction; rupture or thinning of the posterior capsule, giving rise to hernia of the lens; enlargement of the lens; displacement of the nucleus, and Reese's theory of some inflammatory or irritative process present on the embryonal nucleus at the time the adult nucleus begins to form, which causes a local increased growth of the posterior capsule of the lens, which is in direct apposition with the embryonal nucleus, and results in a relatively greater growth of the adult nucleus at this point. The slits and empty spaces which are sometimes seen from the embryonal nucleus to the posterior capsule are thought by Reese[32] to be the result of the rapid growth of the adult nucleus, and are more numerous posteriorly because the

31. Tyson, H. H.: Lenticonus Posterior, Arch. Ophth. **57**:38 (Jan.) 1928.

32. Reese, W. S.: Tr. Am. Ophth. Soc. **26**:339, 1928.

33. Butler, T. H.: Lenticonus Posterior: Report of Six Cases, Arch. Ophth. **3**:425 (April) 1930.

process began slightly before the production of the lens fibers in the adult nucleus had time to progress very far. However, Reese thinks that these spaces become less numerous as the distance between the posterior capsule and the embryonal nucleus increases and the adult nucleus enlarges.

Marsh [34] recently brought the literature up to date on this subject and advanced the theory of "accommodative strain." So far no microscopic studies have been made of an eye in which posterior lenticonus or lentiglobus has been diagnosed previous to enucleation, and as yet no theory explaining the cause of this lenticular condition has been satisfactorily proved.

COMMENT

I believe this case to be one of coloboma of the optic nerve not involving the choroid; however, it may be open to dispute owing to the irregular distribution of choroidal pigment on the temporal side. Nevertheless, the margins were so well defined and the excavation so marked that it is hard to conceive of the choroid not being depressed and gradually sloping into the excavation if it were really involved in the coloboma.

The vessels seemed to emerge from the lower portion of the disk, and those to the upper part of the fundus turned before they came into view near the center. The lower vessels gave the appearance of coming from outside the nerve, and I think that the vascular arrangement can be placed in Caspar's first classification.

The size of the pseudodisk was about twice the normal, and it was grayish white. The depth was hard to estimate due to the pathologic process in the posterior portion of the lens, but certainly it was 10 D. or more and the inferior portion was deeper than the upper portion.

The posterior lenticonus gave all the characteristic appearance ophthalmoscopically, and was definitely seen with the slit lamp. This diagnosis was verified by Dr. Cowan. The lenticonus was unilateral, and opacities and vacuoles were seen in the posterior portion, as described by Reese. There was a large strand of what looked to be hyaloid membrane adherent to the posterior capsule at its vertex.

The fact that in this case a coloboma of the optic nerve was present associated with a posterior lenticonus leads one to wonder about the relation of the cause of the two. Is it not possible that the union of mesoblast which prevents the closure of the fetal cleft at its posterior tip, thereby causing a coloboma of the optic nerve, might not extend forward through the vitreous from the pseudodisk to the poste-

34. Marsh, E. J.: Lenticonus Posterior: Further Study, Arch. Ophth. **8**:804 (Dec.) 1932.

rior fibrovascular sheath about the lens, and cause an undue tension backward, giving rise to a posterior bulging of the structure of the lens itself, namely, a posterior lenticonus? Or, if one accepts Kirby's [35] belief that the remains of the hyaloid vessels are attached to the anterior condensation layer of the vitreous and not to the capsule of the lens, would it not be possible for the patellar fossa to be deepened by this contraction, and lead to a backward bulging of the lens? Certainly the association of these two congenital anomalies in the same eye makes one wonder, especially when a large strand of hyaloid membrane is present.

Dr. Arthur J. Bedell contributed the photographs of this patient's fundus and Dr. Alfred Cowan, the slit lamp study and description.

35. Kirby: Tr. Am. Ophth. Soc. **29**:179, 1931.

Obituaries

P. N. K. SCHWENK, M.D.

1854–1934

Dr. P. N. K. Schwenk, of Philadelphia, died on Saturday afternoon, Feb. 17, 1934, following a week's illness from pneumonia.

He was born in Valley View, Pa., on Aug. 24, 1854, and was the son of Abraham and Catharine (Klinger) Schwenk. He was educated in Lewisburg High School and graduated from Buckenell University in 1876. He entered the Medical School of the University of Pennsylvania in 1879, graduating in 1882. He took up the specialty of ophthalmology and was attending surgeon to the eye and ear department of the Pennsylvania Hospital from 1890 to 1915, and attending surgeon to the Wills Hospital from 1897 to 1924, being made consulting surgeon on his retirement in 1924.

He was a member of the Philadelphia County Medical Society, the American Medical Association, the American Ophthalmological Society, the American Academy of Ophthalmology and Oto-Laryngology, the Congress of American Physicians and Surgeons, the College of Physicians of Philadelphia, and the American College of Surgeons. He was a prominent Mason. He is survived by his widow and one son.

The esteem in which Dr. Schwenk was held by his professional confrères, his associates and his friends attests to his noble character and his beautiful life, which is an epic of loyalty to God, to family and to friends. The world is better that he lived in it, and in the days to come many will miss his advice and companionship. He was spared a long illness, being seriously ill for only a few days. When the end approached, "he went unresistantly, resignedly and fearlessly, like the mariner who, when duty calls, unfurls his sail against the stoutest breeze."

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

ZONULA CILIARIS. JULIO MORENO, Arch. de oftal. hispano-am. 33: 235 (April) 1933.

This is an elaborate study of the zonula from historical, anatomic, histologic, embryologic and biomicroscopic points of view, based on original investigations and finely illustrated with colored plates and black and white figures. The author concludes: 1. The zonular ligament is constituted not of independent fibrils but of a number of fine fibrils joined by an amorphous pellicle constituting an actual membrane. 2. Although it is not yet possible to determine precisely the embryologic origin of the zonula, it seems to be of ectodermal origin and to arise from the cells of the pars ciliaris retinae. 3. The lenticular insertion of the zonula takes place by expansions of the zonular fibers, giving rise to the formation of the so-called zonular lamellae. 4. The zonular ligament constitutes the posterior limit of the posterior chamber. 5. Kuhnt's recessus camerae posterioris terminates in blind pouches at the level of the outer third of the ciliary processes and not immediately in front of the ora serrata as hitherto described. 6. Between the zonula and the hyaloid membrane there is contact, without any continuity of tissue. 7. The best coloring matter for demonstrating the zonula is Ziehl's fuchsin in a 1:3 dilution. This membrane presents no orifices or fissures. 9. An injection can be made into Hanover's space, which forms a real perilenticular canal. 10. The injection of fuchsin into Hanover's space is the best method of staining the zonular fibers simultaneously. 11. It is rare to find a normal zonula by biomicroscopic examination owing to the fact that disease is present in most of the patients examined. 12. The amorphous interfibrillar pellicle is not demonstrated by biomicroscopy.

C. E. FINLAY.

Bacteriology and Serology

THE PATHOGENIC SIGNIFICANCE OF BACILLUS SUBTILIS FOR THE EYE. R. HOFFMAN, Ztschr. f. Augenh. 80: 124 (April) 1933.

The author reports the case of a 23 year old man who received an injury from a small stone striking the eye. There was a perforating scleral wound followed by panophthalmitis and ring abscess of the cornea. Bacteriologic examination showed the presence in the eye of a bacillus of the subtilis group as the probable cause of the inflammation. The author reviews the literature relative to the pathogenic significance of Bacillus subtilis, cites his results in certain experiments on animals and concludes that this usually harmless saphrophyte, while rarely pathogenic for the external eye (corneal ulcer), is more frequently so for the inner eye. When it enters the vitreous it produces an acute inflammation with the usual picture of abscess of the vitreous,

and eventually panophthalmitis. Occasionally a ring abscess of the cornea forms. Experiments with animals show results similar to those in the human eye, except that healing with shrinking of the globe results in the former rather than rupture.

H. L. BEGLE.

Color Sense

FALSE SENSATION OF RELIEF PRODUCED BY LARGE COLOR CONTRASTS. J. ARJONA, Arch. de oftal. hispano-am. **33**: 369 (June) 1933.

This paper deals with the viewing, in binocular vision, of letters or figures in colors of widely different wavelengths at different levels and under certain favorable circumstances. This phenomenon, which was first noted by Brücke and Hering, has been discussed and variously explained by von Kries, Hess, Einthoven, Luckiesh, Bourdon and Marc Landolt. The author differs from these men and attributes the phenomenon to a false estimate of depth dependent on the value attributed, through habit and experience, by the eye or visual centers to the focusing at different levels, in relation to the retinal surface, of colored rays of different refrangibility, accommodation not coming into play. This phenomenon is observed only when no other circumstances lead to a correct estimate of relief.

C. E. FINLAY.

Congenital Anomalies

THE MORPHOLOGY OF HUMAN MICROPHTHALMOS WITH COLOBOMA: PLATE CONSTRUCTION MODEL AFTER MICROSCOPIC SERIAL SECTIONS. R. H. MERRILL, Klin. Monatsbl. f. Augenh. **90**: 454 (April) 1933.

Microphthalmos, complicated by coloboma, occurs with or without appended cysts which cannot be detected prior to enucleation. The larger the cysts, the smaller is the globe, which may show all the stages from a minute rudimentary eye to anophthalmos. Ophthalmoscopic inspection is impossible in this complication, while the fundus may be observed in cases without orbital cysts, provided the lens is clear. It was one of Merrill's purposes to demonstrate the mechanism of this difference by exact histologic research and by construction of the plate model after microscopic serial sections of the microphthalmic globe of a child, 3 days old. This eye was reduced in size in every dimension; the horizontal diameter of the cornea measured 6 mm.; the optic nerve was very thin and was inserted at the lower half of the posterior portion of the globe. The fetal fissure was open along its entire extension. Retinal or orbital cysts were absent. A. von Szily found these cysts to be secondarily enlarged and to represent everted bags of the retina within the area of the coloboma. The insertion of the optic nerve can be located on the upper (dorsal) portion of the globe in microphthalmos coupled with retinal-orbital cysts, and in the lower (ventral) portion in the absence of the cysts. Merrill's research showed that both types represent the terminal result of the same malformation, notwithstanding the different aspect. The difference is brought on by eversion and secondary ectasia of the duplicature of the retina, leading to the forma-

tion of orbital cysts, in the one type, and by the direction of the same process toward the interior of the eye, in the other type. The reasons for this mechanism could not be found. It may be due to primary variations of the anlage, especially the differentiation between the retina and the pigment epithelium, and the rôle that they play in the initial formation of the optic cup. The tissues of the anlage forming the margin of the optic cup turn inward in the one instance and outward in the other. Therefore, it is justifiable to consider the two forms of microphthalmos as one genetic entity with the morphologic distinction between microphthalmos with external, or orbital, cysts and microphthalmos with intraocular cysts.

G. B. HEIDELMANN.

Cornea and Sclera

MOOREN'S ULCER. A. J. BEDELL, Am. J. Ophth. **16:** 289 (April) 1933.

The following summary is given by the author: "A supplementary account of a previously reported case of Mooren's ulcer is recorded by stereoscopic photographs and clinical description.

"Another case of Mooren's ulcer is cited in which it was necessary to enucleate both eyes. There was perforation of the cornea and pathologically the appearance of tuberculosis. Stereoscopic photographs illustrate the unrelenting progress of the disease, which no treatment seemed to control or even retard.

"Intense pain has been spoken of as an outstanding symptom by most observers. In the second case this was almost constant in contrast to the first where the patient never complained of pain.

It would seem from a study of the literature that many cases have been called Mooren's ulcer when they probably belonged in some other group. Actual recovery from a Mooren's ulcer must indeed be rare."

W. S. REESE.

TREATMENT OF CORNEAL INFECTIONS. J. E. WEEKS, Am. J. Ophth. **16:** 293 (April) 1933.

The author suggests in cases of corneal ulcer that toxic processes and foci of infection first be eliminated, and that the patient's general condition be improved by the addition of necessary vitamins to the diet or other general measures. He remarks that most serious corneal infections occur after the age of 40, and that rational treatment is based on the knowledge of the micro-organism present. The thermal ranges of growth of different organisms are enumerated, and attention is called to the consequent futility of hot and cold compresses as germicides or growth-retarding agents. Attention is called to dacryocystitis as a concomitant of corneal ulceration and to the necessity of determining the ocular tension. If increased, it should be lowered by a miotic, if iritis is not present, or by opening the anterior chamber. If the latter is done and hypopyon is present, it should be removed. Atropine should not be used indiscriminately. Various cleansing and germicidal solutions are mentioned, and the author gives his method of treatment of several specific conditions. This interesting article must be read to be appreciated.

W. S. REESE.

NODULAR DEGENERATION OF THE CORNEA. J. H. JUDD, Am. J. Ophth. **16:** 310 (April) 1933.

The author gives the following summary: "1. Nine cases of nodular degeneration of the cornea in three generations are reported with descriptions of the appearance with slitlamp microscopy. 2. The direct descendants of the patient in Case 1 are all very slender and tall, suggesting a pituitary dysfunction. 3. Age of onset varies even in the same sex: Case 2, middle age; Case 9, adolescence. 4. The nodular and latticelike degenerations are both present in this series. These should be considered different manifestations of the same process. 5. The earliest stage can be detected only by slitlamp microscopy (Case 5). 6. Theories of pathogenesis are presented and reports of pathological findings are summarized. 7. Treatment is still unsatisfactory."

W. S. REESE.

CONGENITAL HYALINE MEMBRANES ON THE POSTERIOR SURFACE OF THE CORNEA. I. MANN, Brit. J. Ophth. **17:** 449 (Aug.) 1933.

The author has examined five eyes presenting a hyaline membrane on the posterior surface of the cornea. The first case was that of a man, aged 29. Vision in the right eye with a compound myopic astigmatic correction was 6/6—, and in the left eye with a compound hyperopic astigmatic correction, 6/4. The tension was normal in each eye, and the disks were not cupped. The right eye appeared normal, except that the lesser circle was extremely near the margin of the pupil, and there were a few tags of vascular remnants attached to it.

In the left eye the cornea was slightly smaller than that of the right eye. The central portion of the cornea was normal. In the periphery, however, a semitransparent hyaline membrane appeared applied to the posterior surface of the cornea almost throughout its whole circumference. The inner edge of the membrane formed roughly a circle concentric with the margin of the cornea and lying at about a fourth of the distance from the limbus to the center of the cornea from it. From this inner margin, the membrane extended outward into the angle practically the whole way round, a small gap (roughly between one-seventh and one-eighth of the circumference) occurring in its peripheral part (though not in its inner edge) at 10 o'clock. This membrane was applied firmly to the posterior surface of the cornea around most of the circumference. In the lower part, however, it stood away from the cornea (except at its inner edge) for a short distance, forming in section a cord to the arc of the corneal nerve. It had a slightly granular texture and was imperfectly transparent. The inner edge was marked by irregular small masses of pigmented tissue (pale golden brown) similar to the pigmented tissue composing the anterior layer of the iris. The pattern of the stroma of the iris could be faintly seen through the hyaline membrane and appeared in places (especially the lower nasal side) to be irregular and fluffy, the normal radial appearance being lost. In some places, chiefly immediately below, the peripheral parts of the superficial radial vessels of the iris came forward and adhered to the deep surface of the hyaline membrane.

The superficial layer of the stroma of the iris was composed of abnormally thick, coarse and sparse golden brown strands, which represented a much thickened peripheral part of the pupillary membrane. They extended to the margin of the pupil and would have overlapped it if the pupil had been dilated. Through the gaps between these strands the deep layer of the stroma could be seen. This was paler and grayish, and rather thinner than normal in that the sphincter could be easily seen through it.

The uveal border at the margin of the pupil was abnormally thick and much puckered. From its upper portion a curious, small, fluffy, whitish mass with a few grains of pigment on its surface hung free by a narrow pedicle and just overlapped the edge of the pupil. It resembled a little mass of organized exudate, but since no other inflammatory signs were present and the mass had no adhesion to the capsule of the lens, it also was probably of congenital origin and represented remains of connective tissue. It could not be true pupillary membrane since it was not continuous with the lesser circle, although, since this lay abnormally near the margin of the pupil, it is possible that it represented a displaced portion.

The second case was that of a girl, aged 8 years. The refraction of each eye was compound myopic astigmatism. Vision equaled 6/36 in each eye. The findings were similar to those in the first case.

In Ballantyne's case, which is also described, there were congenital anomalies of the lids and iris with conjunctival dermoids and subconjunctival lipoma.

Two doubtful cases are added—one by Stephenson and one by Coats.

The condition is congenital, and it is in all probability due to an abnormality of the postendothelial tissue occurring at and after the 12 mm. stage. The initial cause of the aberration and also the reason for its assumption of the peculiar appearance described must remain obscure, although the possibility of fetal inflammation acting as the initial cause must be admitted. The article is illustrated.

W. ZENTMAYER.

KAYSER-FLEISCHER'S RING IN CASES OF PSEUDOSCLEROSIS (WILSON'S DISEASE) AMONG THE CHINESE. W. P. LING, Nat. M. J. China **18:** 962 (Oct.) 1932.

For the first time since 1921, when the hospital from which this report appeared was opened, cases of pseudosclerosis came under observation, all four of which showed a typical Kayser-Fleischer ring in the cornea. The patients were men under 30 years of age. In three of them neurologic symptoms characteristic of pseudosclerosis had developed, while the fourth one had not yet shown such symptoms, but had a typical Kayser-Fleischer corneal ring. Two of the patients had a positive family history in that a typical ring was found in some of their brothers. In three of the patients the fundus showed peculiar changes which are being separately dealt with by Pillat. In three of them the dark adaptation was below normal. No "copper cataract" was found. The diagnostic value of Kayser-Fleischer's ring in relation to pseudosclerosis is emphasized.

S. H. MCKEE.

Experimental Pathology

EXPERIMENTAL HYPERTENSION AND RETINAL ARTERIAL SPASM. S. A. AGATSTON, Am. J. Ophth. **16:** 327 (April) 1933.

The author reviews the literature, summarizing it as follows: "1. Spasms of the visceral and cerebral arterioles cause hypertension. 2. Spasms are caused by increased secretion of adrenalin. 3. Stimulation of the depressor nerve of the vagus causes a diminished adrenalin secretion and hypotension. 4. A paralyzing effect on the depressor causes increased secretion of adrenalin and hypertension. 5. This paralyzing effect on the depressor nerve may be caused by a disturbance of the normal function of the thyroid, pancreatic, and pituitary glands. 6. Some change in the blood chemistry or the presence of certain toxins may cause the underlying endocrine disturbance producing hypertension. 7. 'Vagotonine,' a hormone extracted from insulin, has a hypotensive action."

The author produced spasms of the retinal arteries in rabbits by the injection of fecal extract and blood serum from patients with active hypertension.

W. S. REESE.

ACTION OF LEUKOCYTIC TREPHONES ON THE CICATRIZATION OF CORNEAL WOUNDS. O. BARATTA, Rassegna ital. d'ottal. **2:** 105 (Jan.-Feb.) 1933.

The literature on trephones, first prepared by Carrel and Ebeling, and the one ophthalmologic observation on them by Tallei are discussed. The author prepared a suspension of leukocytes by injecting sodium aleuronat into the peritoneal cavity of guinea-pigs, removing the sterile exudate, centrifugating it and washing the sediment. Identical incisions were made in both corneas of rabbits, one eye being treated by instillations of the leukocytic suspension twice a day for twelve days and on alternate days from the twelfth to the twentieth day. Eyes were removed and sectioned after four, eight, twenty-four and thirty hours and after three, four, six, nine and twenty days. In the eyes removed during the first few days, differences between the treated and untreated eyes were noted, the treated eyes showing a more active proliferation of the epithelium and stroma cells bordering the wound. After twenty days no differences were observed. A bibliography is appended.

S. R. GIFFORD.

SARCOMA IN RATS, DEVELOPED THROUGH ABNORMALLY LARGE DOSES OF ULTRAVIOLET RADIATION. K. HULDSCHINSKY, Deutsche med. Wchnschr. **59:** 530 (April 7) 1933.

Two types of lamps producing ultraviolet radiations were used on white rats from 6 to 8 weeks old. The quartz lamp gave off rays under 270 millimicrons while the solar lamp did not. The former was used at 75 cm. for two hours; the latter, at 35 cm. for three hours. In both series the radiation was applied for about a year on five days of the week. Five animals were subjected to the quartz lamp and seven to the solar lamp. All five subjected to the quartz lamp had ocular sarcoma. The dosage was estimated as being ten times that known as the therapeutic dose.

L. L. MAYER.

General

NASAL NERVE SYNDROME. A. DENTI, Rassegna ital. d'ottal. **2:** 32 (Jan.-Feb.) 1933.

The distribution of the nasal nerve, which supplies sensory branches to the globe through the ciliary ganglion and long ciliary nerves and to the sphenoid, ethmoid and nasal mucosa, is described. Attacks of pain with superficial keratitis, cyclitis and iritis, associated with swelling and congestion of the nasal mucosa and relieved by cocainization of the anterior nasal mucosa, have been described by Charlin and others as the nasal nerve syndrome.

The author reports two cases which he considers belong in this group. In one case a youth of 17 years was subject to attacks of pain and lacrimation of the left eye associated with rhinorrhea on the left side for a year preceding examination. He was seen by the author in an acute attack of cyclitis in the left eye with left-sided rhinorrhea. Vision was reduced to 1/2. The sinuses were normal. After two applications of cocaine and epinephrine to the anterior nasal mucosa the attack subsided promptly. Vision one week later was 10/10, and there were no further attacks for three months. In the second case a woman, aged 20, had attacks of left-sided pain and swelling of the lids from three to four days before menstruation. During such an attack the cornea and media were unaffected, the pain and a marked edema of conjunctiva and lids being the only ocular findings. The nasal findings resembled those in the previous case. Three nasal applications of cocaine and epinephrine relieved the symptoms, which did not recur during the following three months.

S. R. GIFFORD.

A NEW PROSTHESIS TO PREVENT LESIONS OF THE EYES BY ROENTGEN RAYS. E. WÖLFFLIN, Klin. Monatsbl. f. Augenh. **89:** 214 (Aug.) 1932.

In his first publication (*Klin. Monatsbl. f. Augenh.* **82:** 813, 1929) the author pointed out some disadvantages of his prosthesis against lesions caused by roentgen rays. These disadvantages have been overcome. The improved prosthesis consists of lead, 1.3 mm. thick, covered by galvanization with a very thin layer of cadmium, on which a cover of nickel is placed, also by galvanization. Secondary rays are almost entirely eliminated by this combination, which permits the use of the roentgen rays without resulting irritation of the interior or exterior surfaces of the lids. Rabbits' ears, exposed to very strong doses, showed no effect from secondary rays. The prosthesis, weighing only 10 Gm., is well tolerated by the patient's eyes for longer periods of time. It can be easily sterilized, does not break as does the former prosthesis of glass and may be perforated for local treatment of tumors of the iris.

K. L. STOLL.

General Diseases

VACCINIA OF THE EYELIDS AND CONJUNCTIVA. M. L. FOLK and E. L. TAUBE, Am. J. Ophth. **16:** 36 (Jan.) 1933.

The authors discuss vaccinia of the eyelids and conjunctiva as described in the literature and give their personal observations, with

the following summary and conclusions: 1. A typical case of vaccinia of the eyelids and conjunctiva is reported. 2. This case brings the total number of reported cases to ninety-nine. 3. Experimental work on the methods of inoculation shows that mere contact with the eyelids is sufficient to bring about vaccinia blepharitis, but that for vaccinia of the skin of the lids to develop previous abrasion is necessary. 4. Pathology, immunology, diagnosis, prognosis and treatment are discussed. 5. It is suggested that the possibility of inoculation of the eye with vaccine be more forcibly brought to the attention of physicians, nurses, public health agencies and lay persons in order that proper precautions may be instituted in the handling of the vaccination wound or the vaccine lymph.

W. S. REESE.

ENDARTERITIS OBLITERANS WITH SPONTANEOUS GANGRENE OF BOTH CORNEAE. A. O. PFINGST and C. D. TOWNES, Am. J. Ophth. **16:** 39 (Jan.) 1933.

The authors review the literature on Buerger's disease and the other interesting diseases of the peripheral arteries. The pathology and etiology particularly are discussed. The case of a white girl, aged 9, a gentile, is reported. She was first seen on Dec. 26, 1930, because of marked protrusion and swelling of the lids of the left eye which had begun six days previously, with what appeared to be a large subconjunctival hemorrhage. The condition became more marked and rapidly developed into a hematoma which soon covered the cornea and protruded between the lids. The child previously had tonsillitis, an aural disturbance and transient facial palsy. She also had had chorioretinitis and, notwithstanding a negative Wassermann test, had been given neoarsphenamine and potassium iodide. The family history was unimportant, and the results of physical examination were negative. An exploratory operation was done, and the cornea was found to be absent, the sclera being filled with clotted blood. During the operation a spinal puncture was made, but the fluid also showed a negative Wassermann reaction. On February 7, the right eye presented a subconjunctival hematoma similar to that of the left eye. The patient died on March 12. The following summary of the results of autopsy is given: "A general and widespread productive endarteritis with no evidence of inflammatory infiltration anywhere with secondary destruction of one kidney, the formation of a brain cyst and the destruction of the corneae of both eyes. Cause of death, a terminal pneumococcus meningitis most likely of otitic origin, coincident to the blood vessel changes but having no direct causative relation to it."

W. S. REESE.

PREMONITORY LID EDEMA IN THE TYPHOID GROUP. S. J. BEACH, Am. J. Ophth. **16:** 119 (Feb.) 1933.

The author reports the case of a woman, aged 22, who for a few days had a symmetrical swelling of the lower lids extending to the cheeks below the zygoma. There was no tenderness or redness. Two days later there was fever, and subsequently the spleen became enlarged. The paratyphoid agglutination test was positive. A second similar case is reported in which Bacillus typhosus was found and a positive Widal

test was obtained. The author speculates as to whether a special strain of typhoid bacilli is prone to cause these swellings of the lids and as to whether, when the swelling is present, typhoid does not have a less severe course.

W. S. REESE.

A CASE OF LEPROSY OF BOTH EYES. M. BOEMER, Klin. Monatsbl. f. Augenh. **88:** 329 (March) 1932.

A merchant who had lived in Dutch Guiana since 1914 had lost weight and been fatigued since 1926. In 1927 he noticed a brownish-red spot on his elbow, which was recognized as lepra. He returned to Germany soon afterward, but did not consult a physician until 1930, when large ulcers on his arms and legs and facies leontina developed. These symptoms disappeared to a large extent under continuous treatment with potassium iodide, iodized sesame oil 40 per cent and especially an ester of oil of chaulmoogra with 1 per cent camphor. Both eyes were normal at that time, with the exception of scleritis. Severe iritis, scleritis, episcleritis and sclerosis of the cornea developed a year later. Lepra bacilli were found. Treatment with gold preparations, as well as with oil of chaulmoogra, was given; local treatment of the eyes was also employed, including cauterization and incision, which resulted in the clearing up of the nodules on the sclera and of the inflammation. No relapse had been observed since October, 1931. The eyes were normal, the general condition was improved. Antileprous treatment was continued, although the case was pronounced not contagious by experts on tropical diseases.

K. L. STOLL.

GRIPPAL DISEASES OF THE EYE AND THEIR PATHOGENESIS. M. A. DVORJETZ, Russk. arch. oftal. **8:** 841, 1932.

The author believes that grip plays an important part in the pathogenesis of various ocular diseases, which occurred, on the average, in 25 per cent of the cases during the recent epidemic of grip, according to the experience of the author and reports in the literature. The author collected his material during the epidemics of influenza in the years 1927-1928 and 1930-1931. There were a number of cases of catarrhal conjunctivitis; twelve of them were rather severe, marginal corneal ulcers developing in five. Two cases of herpes of the cornea, two of deep keratitis, two of disciform keratitis and several of phlyctenular keratitis demonstrate the ease with which the cornea is affected. There were two rather unusual cases of punctate hemorrhages into the scleral tissue. Dvorjetz observed several cases of neuralgia of the supra-orbital nerve, four cases of retrobulbar neuritis, three of choked disk and one of hemorrhagic retinitis. The peculiar feature was that two definite groups of serious diseases of the eye were observed during each epidemic. The uveal tract was affected widely during the year 1927-1928, as there were twelve cases of plastic iridocyclitis and seven of metastatic uveitis, while in the 1930-1931 epidemic disease of the orbit was observed instead. The cases of plastic iritis occurred during the period of convalescence and lasted from four to six weeks; recovery was complete. The cases of metastatic uveitis occurred mostly in chil-

dren; all led to loss of sight, phthisis bulbi being the end-result. Five cases of orbital cellulitis were primary, and in seven patients this condition was secondary to infection of the nasal cavities. In instances the orbital disease subsided under conservative or operative treatment of the sinuses involved.

The fact that two different parts of the visual organ were affected during the two epidemics is ascribed to the difference in the micro-organisms causing the influenza.

O. SITCHEVSKA.

Glaucoma

DESTRUCTION OF BLOOD VESSELS IN JUVENILE GLAUCOMA. B. G. TOWBIN and L. I. WILENSKI, Ztschr. f. Augenh. **80:** 141 (April) 1933.

Glaucoma in young persons occurs in two forms: (1) that in which the size of the globe increases, hydrophthalmos, and (2) that which clinically resembles the glaucoma of older persons, or juvenile glaucoma. During a period of ten years there were observed in the ophthalmic clinic of the I. Moscow State University 784 cases of glaucoma with the disease occurring in 12 persons not over 30 years of age. In the ophthalmic clinic of the Kasan State Institute, among 256 cases of glaucoma there were 14 in which the patient was under 30 years of age—5 with hydrophthalmos and 7 with juvenile glaucoma. The authors believe that every patient with juvenile glaucoma should have a careful general examination, for departures from the normal may be found which may prove significant from the standpoint of causation. They cite their findings in an extensive examination of a patient with juvenile glaucoma who showed changes in the vascular system: 1. The capillaries of the skin at the bed of the finger-nails were examined with the Leitz capillaroscope. There were changes in the capillary network, narrowing of the arterial branches, stasis, dilatation of the venous branches and corkscrew vessels. 2. There was a moderate increase in the blood pressure. 3. There were changes in the concentration and secretion of the urine.

H. L. BEGLE.

Injuries

EYELASHES IN THE ANTERIOR CHAMBER. A. M. MOVSHOVITZ, Russk. arch. oftal. **8:** 899, 1932.

A butcher had his right eye injured with a piece of bone while he was cutting meat. On examination the eyeball showed marked inflammation with a linear wound on the cornea about 5 o'clock. The iris was prolapsed into the wound. There were two lashes on the iris forming a sharp angle with each other at 1 o'clock. The lens and vitreous were clear; vision was 0.3. No foreign body was seen in the roentgenogram. The prolapsed iris was cut off, but the eyelashes were left in the anterior chamber, as the author preferred not to enlarge the wound. The inflammation of the eyeball subsided within two weeks, and vision returned to normal.

O. SITCHEVSKA.

Lens

AMBARD'S CONSTANT IN SENILE CATARACT. M. VELE, Rassegna ital. d'ottal. **2:** 88 (Jan.-Feb.) 1933.

The relation between the urea in the blood and that in the urine (Ambard's constant) was determined in twenty cataractous and twenty normal persons of about the same age. In normal persons above 45 years of age the constant was about 0.075, and in persons with cataract it varied from 0.074 to 0.17, showing no constant variation from the normal. The author concludes that changes in renal permeability are no greater in persons with cataract than in normal persons of the same age. A bibliography is included.

S. R. GIFFORD.

PECULIAR FORM OF CATARACT AFTER ALLEGED INJURY, PERHAPS SIDEROSIS OF THE LENS. A. BALCAREK, Klin. Monatsbl. f. Augenh. **88:** 332 (March) 1932.

A man, aged 20, fell 5 meters from a roof eighteen months prior to his admission to the hospital. No lesion of the eyes was noticed at that time, but vison began to fail in the left eye a year after the accident. A fine superficial opacity in the pupillary area of the cornea and a cataract were found. No intra-ocular foreign body could be detected by roentgenologic or other examinations. After full mydriasis a circle of yellowish-brown pigment spots of equal size were noticed, arranged at equal distances from one another and corresponding to the seam of the dilated pupil. They were located directly below the anterior capsule. The surface of the lens showed no discontinuation. The presence of siderosis of the lens could not be established in this case, as was possible in another case reported by the author, in which a small iron splinter had lodged in the lens. Convinced that the cataract was of traumatic origin, Balcarek considers whether the pigment spots represent xenogenous or hematogenous siderosis. He leaves this question, as well as the fixation of the time at which the cataract may have formed, unsettled.

K. L. STOLL.

Methods of Examination

AN IMPROVED AND A NEW TEST FOR STEREOSCOPIC VISION. F. H. VERHOEFF, Am. J. Ophth. **16:** 589 (July) 1933.

The author refers to a previous article in which he described a test for stereoscopic vision based not on the effect of the perception of depth but on the fact that (1) two disparate retinal images are "fused" in the brain into a single intermediate image only in those with the faculty of stereoscopic vision, and (2) one retinal image in one eye can in the brain apparently fuse with and alter the position of two images from the other eye, each of which is slightly disparate with regard to it. The new test is based on the perception of luster and also on the fact that if two nearly vertical black lines on a white background, at a slight angle to each other, are fused by means of a stereoscope, they appear as a single intermediate line with a fore-and-aft inclination. The new test is described and illustrated. The paper must be read to be appreciated.

W. S. REESE.

A Simple Technic for Roentgenography of the Optic Canal. A. Druckmann, Folia ophth. orient. **2:** 208, 1933.

Asserting that the Goalwin technic for roentgenography of the optic canal is too complicated, the author has devised a simpler method. The patient lies prone, with the forehead and nose touching the plate. The tube is inclined to form an angle of 40 degrees, open forward, with the horizontal plane, and rotated sideways to form an angle of 30 degrees, open upward, with the sagittal plane. The central ray is then directed toward the center of the orbital opening. To the users of this method the author "guarantees a certain and satisfactory picture."

G. M. Bruce.

Neurology

Retinal Circulatory Disturbances in Epilepsy. G. Rossi, Arch. di ottal. **39:** 502 (Nov.-Dec.) 1932.

Using Bailliart's dynamometer, the author noted a hypertension in the retinal arteries during epileptic attacks, with no abnormalities, as a rule, during intervals of freedom from attacks. He gives the details of two cases followed with special care. The first showed hyperemia of the fundus during free intervals, with marked ischemia during attacks. The fields were contracted, and vision was reduced to 1/6 in each eye. The second case also showed venous stasis with blurred outlines of the disks during the free intervals and just after the attacks. The blood pressure was slightly below normal (100 systolic and 70 diastolic), but the retinal vessels showed a definite hypertension, averaging from 45 to 50 mm. of mercury (diastolic). No records could be made during the brief attacks. These findings suggest a constantly increased intracranial pressure with marked angiospasm during the attacks, in one case probably involving the cerebral vessels. A bibliography is appended.

S. R. Gifford.

Casuistic Contribution to the Rarity of Dystrophia Myotonica in Switzerland. W. Tschopp, Schweiz. med. Wchnschr. **63:** 657 (July 8) 1933.

Rougues' monograph has demonstrated the incidence of myotonic dystrophy in all of Europe and North America. Greenfield's review in 1923 showed over two hundred cases reported in the literature. Cases have been reported from Switzerland by Vogt, Lüssi and H. C. Frey. The author adds three cases after a brief but fairly complete review of the literature of the subject. Of interest to the ophthalmologist are the changes in the lens which are of such a characteristic nature and so often a part of this disease. One patient had an extra-ocular muscle affected, causing diplopia, which has been reported only once previously by Breidenbach. Together with the other characteristic signs of the disease, the cataract of dystrophia myotonica, so thoroughly described by Vogt with the aid of the slit lamp, beginning in adolescence was noted in all the author's cases. A lengthy bibliography is appended.

L. L. Mayer.

BLINDNESS PRODUCED BY BILATERAL MICROGYRIA IN THE CALCARINE REGION. L. TOKAY, Arch. f. Ophth. **129:** 426 (Jan.) 1933.

A youth, aged 20, who had been blind since early childhood and epileptic since the age of 5, died of pneumonia. Autopsy revealed, in addition to many other anomalies, microgyria of both calcarine regions. The optic nerves were normal, but the lateral geniculate bodies were completely atrophic and replaced by vicarious glial proliferation. This is explained as retrograde atrophy from inactivity after destruction of the area striata.

P. C. KRONFELD.

Ocular Muscles

THE TREATMENT OF NONPARALYTIC SQUINT. L. C. PETER, Am. J. Ophth. **16:** 481 (June) 1933.

The author stresses the importance of the early treatment of squint. He takes up first monolateral esotropia and briefly discusses its etiology. Treatment is discussed from the standpoint of refraction, amblyopia, training the fusion faculty, surgical intervention and stereoscopic training. Alternating esotropia and divergent squint are similarly dealt with.

W. S. REESE.

Orbit, Eyeball and Accessory Sinuses

ECHINOCOCCUS IN THE ORBIT AND TRAUMA: REPORT OF A CASE. O. A. DUDINOW, Klin. Monatsbl. f. Augenh. **88:** 373 (March) 1932.

Echinococcic disease of the orbit is very rare. One case occurred in Russia among 50,000 ocular cases, according to Markow's statistics. The percentage is still lower in Western Europe. Dudinow reports the case of a boy, aged 11 years, who received a severe blow on his left eye from the hoof of a donkey five years prior to his admission to the hospital. A bluish swelling remained for two months. A year later a small tumor developed near the nasal canthus; it grew slowly, causing no pain but progressive decrease of vision. Examination revealed a round tumor, the size of a pigeon's egg, its upper border well defined from the orbital margin and the overlying skin movable. The tumor extended into the orbit. It showed fluctuation and displaced the globe downward and to the temporal side, causing, also, slight proptosis. The pupil was round and of sluggish reaction, the disk slightly indistinct, and vision greatly reduced. The boy's general physical condition was good, but 9.5 per cent eosinophil cells were found. The tumor, extirpated under local anesthesia, adhered to the bone in the region of the trochlea and to the upper and medial wall of the orbit, extending within and below the neighborhood of the disk. Two cysts were found within the tumor: a so-called Echinococcus cysticus fertilis and an attached secondary cyst. The linings of both cysts were covered with numerous budding cysts, discernible with the naked eye as minute yellow dots. Dudinow believes that the trauma favored the development of the echinococcus in the orbit, analogous to the relations between injuries and tuberculous or syphilitic lesions of the bones.

K. L. STOLL.

Pharmacology

THE ESTERS OF CHOLINE IN OPHTHALMOLOGY. M. VILLARET, L. JUSTIN-BESANÇON, (MME.) SCHIFF-WERTHEIMER and J. GALLOIS, Arch. d'opht. **49:** 129 (March) 1932.

In the first section the chemical composition of this substance and the large number of substitutions possible are discussed. Acetylcholine and α-methylacetylcholine, the two of special interest to ophthalmology, belong to the group of derivatives which have the property of lowering the blood pressure.

In the second section the pharmacologic properties of acetylcholine are discussed. Experiments indicate that when given subcutaneously there is a wide margin between its toxic and its active dose. It lowers the blood pressure by its action on the heart and intense vasodilatation. When injected subcutaneously, it has no action on the heart, although its vasodilating effect remains. Its vasodilating action depends on the arteriolar tone. When this tone is already relaxed, it has no effect. Experiments are presented which seem to indicate that the dilatation of the retinal arteries following subcutaneous administration is not related to the fall in arterial tension. When administered in conjunction with epinephrine, a peripheral dilatation occurs without a fall in blood pressure. It has a powerful miotic action when injected subconjunctivally, but no action when injected subcutaneously. When instilled into the conjunctival sac in sufficient strength, it has a powerful miotic action and constitutes an extremely efficient antagonist to atropine. It has been found in the retina and aqueous in appreciable quantities.

The third section deals with the action of α-methylacetylcholine on the iris and the ocular tension. The racemic form only is considered. The authors have verified the experiments of Reid Hunt, who, however, made no observations on the intra-ocular tension. This drug produces a miosis varying in intensity with the strength used when dropped into the conjunctiva. It has no appreciable effect on the mydriasis produced by atropine. Subconjunctival injection produces a rapid miosis, but its duration is shorter than when instilled. Subcutaneous injection produces the same effect as subconjunctival injection, the miosis being equal on the two sides. When instilled, this drug produces a rapid fall in the intra-ocular tension to about 15 mm., which lasts for about twenty minutes. This hypotony is followed by a secondary rise to the previous level or above it in from sixty to eighty minutes which, in turn, is followed by a secondary hypotension. This cycle is unaffected by the addition of pilocarpine. Subconjunctival administration produces a sudden severe fall lasting one or two minutes followed by a progressive accentuation of this fall, which persists without a secondary rise if the animal lives. An immediate and profound drop in the blood pressure is produced either by subconjunctival or by subcutaneous injection. Because of the intense miosis, the effect on the retinal vessels in rabbits cannot be seen, but some change in the conjunctival circulation has been observed. Salivation and lacrimation are also produced.

The fourth section presents the effect on man of subcutaneous and intramuscular injections of acetylcholine. A dose of 20 cg. produces no vasodilation of the capillaries as shown by flushing of the face.

Ophthalmoscopic observation has, in spite of the possible sources of error, demonstrated a definite dilatation of the central retinal artery quite regularly. An average dose of from 5 to 15 cg. produces only a slight fall in the blood pressure. A study of the effect on the intra-ocular tension has shown that the tension was lowered only in those eyes with a normal tension. In those eyes in which it was not lowered, the tension was in the neighborhood of 25 mm. Their observations indicate that in those eyes with a moderate elevation of tension acetylcholine produces a slight but definite and lasting hypertonic reaction.

In the fifth part of this report, the authors discuss the use of acetylcholine alone in (1) obliteration of the main trunk or branches of the central retinal artery, (2) thrombosis of the central retinal vein, (3) chronic glaucoma, (4) retrobulbar neuritis and (5) visual disturbances of central origin. In dealing with obliteration of the central retinal artery, the authors point out that it is difficult to decide whether the cause is an embolus, a thrombus or spasm of the artery. Acetylcholine can act only in the two latter. They refer to the cases reported in the literature which have been favorably influenced by this drug. According to the reports in the literature, amblyopia caused by quinine has also been benefited. Posthemorrhagic blindness has not so far been helped.

Thrombosis of the central retinal vein has been treated sucessfully in two reported instances.

In chronic glaucoma, acetylcholine used in conjunction with miotics has had a favorable effect on the tension. Because the effects of this drug on intra-ocular tension are not fully understood, it should be used only when careful observation of the intra-ocular tension is possible.

As the pathologic process of retrobulbar neuritis may primarily be of a vascular nature, its use has been suggested in the treatment of this condition. The authors have had no personal experience in such a case.

In regard to cortical blindness—hemianopia—although some cases of recovery following the use of this drug have been reported, the authors point out that any such recovery should not be attributed solely to the action of this drug as sometimes spontaneous cure occurs.

In conclusion, the authors express the belief that the elective action of this drug on the peripheral arteries makes it the medication of choice in those conditions in which vascular spasm plays an important part. This arteriolar dilating action also is useful in the treatment of glaucoma, as the tolerance of the organism permits its prolonged use in the development of a chronic case. Further research on the other derivatives of choline may result in the development of valuable new therapeutic agents.

S. B. MARLOW.

ACTION OF HISTAMINE ON THE NORMAL AND THE GLAUCOMATOUS EYE. B. ALAJMO, Rassegna ital. d'ottal. **2**: 3 (Jan.-Feb.) 1933.

Previous work on histamine, including that by Hamburger, is reviewed. Using from 1 to 5 per cent histamine hydrochloride, the author observed in rabbits after instillation a marked chemosis and hyperemia of the vessels of the iris, accompanied by miosis. The tension was reduced to 5 to 10 mm. Similar, but more marked, effects followed subconjunctival injection. The injection of 0.2 cc. of a 1 : 500

solution into the vitreous produced a marked lowering of tension (below 10 mm.) and, after twelve hours, an intense plastic uveitis. Sections showed an area of infiltration containing large numbers of eosinophil cells. Dropping 2 per cent histamine on the iris of enucleated rabbit eyes from which the cornea had been removed produced marked miosis. Intravenous injections produced a fall of blood pressure and intraocular tension and a definite mydriasis. The same effect was noted in dogs. Instillation of histamine converted the mydriasis produced by cocaine to a slight miosis and brought the pupil dilated by atropine to its normal diameter in fifteen minutes. It increased the miosis produced by pilocarpine and physostigmine.

In the normal human eye the instillation of 3 per cent histamine produced a sensation of burning for three or four minutes, followed by intense congestion and chemosis. The pupil contracted after from eight to ten minutes in some persons, but never more than 1.5 mm., and usually returned to normal after several hours. No influence on the ocular tension was observed.

Instillation of a 3 per cent solution was employed in three cases of acute, four of chronic inflammatory and eight of chronic simple glaucoma. A more marked chemosis occurred in the glaucomatous eyes than in normal eyes, and the pain of acute glaucoma was increased. In two acute cases the pupil remained unchanged, while in the others the effect varied. The miosis, however, when it occurred, was never more than 2 mm. The tension showed, instead of the decrease reported by Hamburger, an increase of about 10 mm. in nearly all cases, reaching 15 and 20 mm., respectively, in two cases of acute glaucoma. It returned to normal in from four to six hours. In only one case of acute glaucoma did a decrease (from 120 to 90 mm.) occur.

The difference between the reaction of normal and of glaucomatous eyes to histamine suggested that histamine-like substances might be present in the blood of glaucomatous patients, but tests of patients' serum injected into dogs and tests of isolated guinea-pig's uterus failed to reveal evidence of this. Negative results were also obtained with the aqueous from two cases of chronic inflammatory glaucoma. These negative results do not exclude the possibility that small amounts of such substances may be present at the onset of an attack of glaucoma, and the author is inclined to believe that acute glaucoma may be a result of local histamine shock. Although he does not so express himself, his results would appear to cast grave doubt on any therapeutic value of histamine.

S. R. GIFFORD.

EXPERIMENTS ON ISOLATED IRIS MUSCLES IN PHARMACOLOGIC RESEARCH. K. VELHAGEN, Arch. f. Augenh. **107**: 172, 1933.

The author describes an instrument for recording the movements of isolated strips of iris when acted on by various drugs. The apparatus previously devised for this purpose is reviewed, and the description of the instrument is sufficient for those interested in duplicating it. Its chief departure from the devices previously used is the recording by shadowgraphs, which does away with the friction of writing levers on the smoked drum.

Curves are given which show the contracting effect of dilute solutions of acetylcholine on the isolated sphincter muscle and the relaxing effect of epinephrine. Acetlycholine is effective in as high a dilution as 1:4,000,000. The effect is abolished by a previous application of atropine.

F. H. ADLER.

RESORPTION OF ATROPINE OINTMENT FROM THE CONJUNCTIVAL SAC. M. KLEIN, Arch. f. Ophth. **129:** 413 (Jan.) 1933.

Just as in the case of sodium iodide ointments, the resorption of atropine in ointment form from the conjunctival sac depends mainly on whether the water in which the atropine is dissolved is the outer or the inner phase in the water-oil emulsion. In the former case, the atropine comes in direct contact with the eye and is readily absorbed. In the latter case, the atropine remains surrounded by a coat of oil and is thus prevented from contact with the cornea. The lowest concentration which produced the smallest noticeable dilation of the pupil in man was 1:200,000 in the case of the oil-water emulsion and 1:10,000 in the case of the water-oil emulsion. The oil-water emulsion is therefore preferable to the other type. At the present time, the only fatty base which gives an oil-water emulsion is lard, the use of which, however, is limited because of its chemical instability.

P. C. KRONFELD.

The Pupil

THE PUPIL IN SEVERE CEREBRAL INSULTS. L. POPPER, Wien. med. Wchnschr. **83:** 120 (Jan. 21) 1933.

In the past ten years, 160 cases of hemorrhage of the brain were studied; in 110 cases the hemorrhage broke through into the ventricle. Immediately after the accident the pupil on the side of the hemorrhage was usually found to be narrower than that on the opposite side. With an increase in the hemorrhage and rupture into the ventricle, the pupil on the side of the hemorrhage became maximally dilated. This condition was usually followed almost immediately by death. The author believed that the dilatation is due to direct stimulation of the sympathetic center in the hypothalamus, as death follows owing to failure of the respiratory center. He believes that for diagnosis and prognosis this is a valuable sign.

L. L. MAYER.

Physiology

THE PLATINUM POTENTIAL AND THE OXIDATION-REDUCTION EQUILIBRIUM OF THE LENS. J. NORDMANN and P. REISS, Compt. rend. Soc. de biol. **112:** 812, 1933.

Through a small puncture in the cornea or sclera, an electrode of extremely thin platino-iridium wire was placed directly on the lens of living, anesthetized dogs, rabbits and guinea-pigs. The potential was then noted in relation to an electrode of mild mercurous chloride placed in contact with the ear. It took from several minutes to half an hour for the potential to develop. The average reading was + 87 millivolts for normal eyes and + 99 millivolts for eyes previously traumatized.

The potential of the lens is less than that of the vitreous and shows more individual variations but is relatively independent of circulatory conditions.

J. E. LEBENSOHN.

THE OXIDATION-REDUCTION BUFFER ABILITY OF THE LENS. J. NORDMANN, Compt. rend. Soc. de biol. **112:** 1515, 1933.

In order to avoid oxidation from the air, fresh lenses were immediately frozen with carbon dioxide in a pyrex mortar and powdered, and a phosphate buffer at p_H Δ 0.6 (depression of the freezing point), was immediately added. Titration was then done with a Vellenger electrode in an atmosphere of nitrogen. Sodium hydrosulphite was used as a reducing agent, and potassium ferricyanide as an oxidizer, and the change of potential was noted in relation to the amount of reagent added. From the resulting curve, Nordmann concludes that the lens can resist oxidative factors but is not able to oppose reducing agents.

J. E. LEBENSOHN.

TONOMETRY AND TONOSCOPY IN PREGNANCY, LABOR AND THE PUERPERIUM. A. FERRARI, Arch. di ottal. **39:** 471 (Nov.-Dec.) 1932.

Previous observations have indicated a slight decrease of intra-ocular tension during pregnancy; this has no relation, as a rule, to changes in blood pressure. The author chose thirty normal pregnant women with normal vision and fundi. On each of this group, three times a day during the period of confinement until ten days post partum, a record was made of the intra-ocular tension, the diastolic pressure in the retinal arteries according to Bailliart and the diastolic and systolic blood pressure in the brachial artery. These observations were carried out during labor in the intervals between uterine contractions.

The intra-ocular tension was found to average 17.3 mm. (Schiötz) during the last weeks of pregnancy, 17.5 mm. during labor and 17.7 mm. in the puerperium—a slight hypotonia if 19 or 20 mm. is taken as normal. No persons were found with an average tension below 16 or above 20 mm. The average diastolic arterial pressure of the retinal arteries was 40 mm. of mercury, and figures slightly below the normal of from 40 to 45 mm. were found in twenty-four during pregnancy, in twenty-three during labor and in twenty-seven during the puerperium. The normal blood pressure underwent no important variations, and the slight reduction in retinal arterial pressure loses any significance when one compares it with the reduction in intra-ocular tension.

Possible causes of the slight ocular hypotension are discussed, the author believing that changes in the blood chemistry, slight acidosis, hyperglycemia and hypercholesteremia must explain it. A bibliography is appended.

S. R. GIFFORD.

Refraction and Accommodation

THE CROSS-CYLINDER TESTS, ESPECIALLY IN RELATION TO THE ASTIGMATIC AXIS. W. H. CRISP, Am. J. Ophth. **15:** 729 (Aug.) 1932.

This article is an exposition of the technic in the use of the cross-cylinder. The value of the cross-cylinder in determining the axis of

astigmatism is especially emphasized. While the author does not recommend that the cross-cylinder shall be used to the practical exclusion of retinoscopy, it is possible to obtain a satisfactory measurement of the refraction at the trial case without any preliminary dependence on retinoscopy. Illustrations and a bibliography accompany the article.

W. ZENTMAYER.

CONTACT GLASSES IN KERATOCONUS AND IN AMETROPIA. O. SITCHEVSKA, Am. J. Ophth. **15:** 1028 (Nov.) 1932.

The article begins with a history of contact glasses in which the characteristics of the Zeiss and Müller glasses are described and their use in keratoconus and ametropia noted.

The author reports on six cases of keratoconus, three in males and three in females. Most of the patients suffered from hypothyroidism. The basal metabolism test gave results within normal limits in all patients. The Abderhalden dialysis tests showed hypofunction of both the thymus and the thyroid glands in one patient. The blood calcium was normal in all, and in none were the cranial bones rarefied. There was no disturbance of the sympathetic nervous system. All had diminution in sensibility of the apex of the cone.

These six patients and one with a high degree of myopia were treated with contact glasses. In five cases the Zeiss contact glasses were tried without success. In two, visual acuity was brought to normal. The myopic patient showed a remarkable improvement with the contact glasses as compared with ordinary lenses. The Müller contact glasses were preferred by the patients as being more comfortable.

W. ZENTMAYER.

Retina and Optic Nerve

OPTIC NEURITIS AND OPTIC ATROPHY DUE TO THALLIUM POISONING FOLLOWING THE PROLONGED USE OF KOREMLU CREAM. G. H. STINE, Am. J. Ophth. **15:** 949 (Oct.) 1932.

Following the prolonged use of Koremlu (a depilatory cream) by a woman, aged 47, polyneuritis, optic neuritis and optic atrophy developed. Relief from sensory symptoms and steady improvement in the motor paralysis followed the withdrawal of the drug and elimination.

Cataract, keratitis, optic neuritis, retrobulbar neuritis and postneuritic atrophy may be caused by thallium.

W. ZENTMAYER.

STUDIES OF RETINOPATHIES: I. DIABETES MELLITUS. E. B. GRESSER, Am. J. Ophth. **16:** 612 (July) 1933.

The author discusses the retinopathy of diabetes. He concludes that the literature indicates that this retinopathy is influenced, if not precipitated, by factors other than the presence of hyperglycemia. The present study covers a hundred cases of known diabetes mellitus. These are of three groups: (1) those characterized by retinopathy; (2) those in which there are sclerotic changes in the retinal vessel alone, and (3) those in which there are no retinal changes. The following summary is given: "In a study of one hundred cases of diabetes mellitus, the clini-

cal signs of hypertension, retinal vascular sclerosis, and abnormal urinary elements are correlated, together with age and duration of the diseased state.

"Thirty-eight per cent of these cases (group 1) presented a retinopathy in which sclerosis and hypertensive signs in the vessels were practically uniformly present. Associated nephritic lesions appeared in 23.5 per cent, of which 10.2 per cent were classed as chronic diffuse glomerular nephritis, 7.7 per cent referred to renal changes in nephrosclerosis. Seventy-two per cent of group 1 were noted as having benign hypertension.

"The average age for this group was 55.13 years and the diabetic state averaged 7.34 years.

"The retinopathy seen in these diabetics did not conform to one clear picture, no entity having been established. The majority showed characteristics of hypertensive retinitis, both vascular and exudative. No relationship was seen between the presence of retinopathy and the severity of diabetes.

"Thirty per cent of the cases presented signs of retinal vascular sclerosis without other vascular or retinal changes. All of these (group 2) were free from renal disease and permanent hyperpiesis was absent. The average age in this group (55.3 years) was practically the same as in group 1, but the duration of the diabetic state was shorter (5.77 years).

"The duration of the diabetic state was not, apparently, a direct factor in the production of retinal pathology.

"The remaining cases (32 per cent, group 3) showed no alterations in the retinal vessels or tissues. Blood pressure ranges were entirely normal and the urinary pictures always negative (sugar excepted). Average age (31.1 years) and duration of diabetes (4.5 years) were lower than in groups 1 and 2. Group 3 was considered the control group."

W. S. REESE.

SOME ASPECTS OF VASCULAR CHANGES IN THE FUNDUS OCULI AND OF RETINAL ARTERIO-SCLEROSIS IN PARTICULAR. P. J. EVANS, Brit. J. Ophth. **17: 257** (May) 1933.

This is the author's thesis for the degree of M.B. (Cambridge). It represents a painstaking study. The cases are tabulated as to age, sex, retinal vascular changes, blood pressure, condition of the radial arteries, heart, urine, other conditions of arteriosclerosis, other conditions of the eye and family history. There are four groups of cases, including observations on almost 200 patients.

The conclusions agree, on the whole, with previously published findings. The author states: "There is reason to believe that certain types of exudate originate primarily in a degenerative lesion of the choroid, to which the retinal changes are secondary. Such changes may occur apart from the changes of retinal arterio-sclerosis. Senile exudative retinitis and retinitis circinata probably belong to this group. The incidence is usually later in life and the prognosis for life surprisingly good.

"The prognostic significance of retinal arterio-sclerosis is very unreliable owing to the frequently localized extent of the lesion. Its presence should, however, arouse caution and emphasize the importance of a careful general examination of the vascular system and of future observation.

"The evidence found by these investigations points to the extremely chronic nature of arterio-sclerosis and to the possibility of its occurrence for many years without causing any gross ill-health, and suggests that, if properly treated, it is not so serious a condition as the literature of recent years would lead one to believe." W. ZENTMAYER.

A SERIES OF THIRTY-ONE CASES OF RETINAL DETACHMENT TREATED BY DIATHERMY. E. F. KING, Brit. J. Ophth. **17**: 287 (May) 1933.

The author gives a brief survey of the operations performed for detachment of the retina at Moorfields Eye Hospital during the last three years and an analysis of 31 cases in which diathermy was used.

Of the first 200 patients treated by Gonin's method, 27.5 per cent were cured; that is, they left the hospital with a full field of vision and no visible detachment.

Of the first 30 patients treated by Guist's method, 45 per cent were cured. In the next 42 cases, there were only 21.4 per cent of cures. In none of the series was selection exercised.

The diathermy method employed was essentially that of Larsson, and the technic is described in full. Of the 31 patients, 18 were cured, or 58.06 per cent.

The following conclusions seem justified:

Diathermy as a method of cauterization for the treatment of retinal detachment is preferable to the thermocautery used by Gonin or the solid potassium hydroxide used by Guist. Further, the technical difficulties are considerably less than in Guist's operation.

Cases in which no hole can be found and those in which a previous operation for detachment, of any type, has been done offer small hope of success.

The age of the patient is of small importance, but the prognosis is much less favorable when the symptoms of detachment are of long standing.

Finally, as might be expected, almost all patients showing inflammatory signs, postcortical changes in the lens, gross opacities of the vitreous and hypotension do badly, while those with a relatively clear vitreous, a discrete hole or holes and normal tension show the most favorable results. W. ZENTMAYER.

THE HISTOLOGICAL APPEARANCES OF AN EYE SUCCESSFULLY TREATED BY DIATHERMY FOR RETINAL DETACHMENT. FATAL TERMINATION FROM PULMONARY THROMBOSIS ON THE NINETEENTH DAY AFTER OPERATION. H. B. STALLARD, Brit. J. Ophth. **17**: 294 (May) 1933.

The patient was a man, aged 74. A modified Larsson diathermy operation had been successfully performed by Doyne. On the thirteenth day a small pulmonary infarct occurred, and on the nineteenth day the patient died of an extensive pulmonary thrombosis.

The eye was removed, and the histologic changes observed led to the following conclusions:

The histologic picture induced by the application of diathermy to the sclera in the treatment of retinal detachment is that of a localized uveitis. Buds of granulation tissue herniating through Bruch's membrane play an important part as the grappling irons that secure the detached retina when it has come alongside of the choroid after the subretinal fluid has been drained. These buds of granulation tissue contain fibroblasts, the precursors of fibrous tissue. Repair is eventually perfected by choroidoretinal fibrous adhesions.

It is evident from this specimen that diathermy applied near the ora serrata is liable to induce a cyclitis, and the amount of fibrous tissue formed in the circumlental space prophesies more remote complications to follow.

W. ZENTMAYER.

THE DOSAGE FOR TRANSCLERAL DIATHERMIC COAGULATION BY MEANS OF A PYROMETRIC ELECTRODE. L. COPPEZ, Arch. d'opht. **50:** 122 (Feb.) 1933.

The author points out that the great defect in all the methods of treatment used for detachment of the retina is lack of control of the amount of heat used. This applies not only to Gonin's procedure but also to transcleral diathermic coagulation as proposed by Larsson. It is especially important in the latter procedure, as the extent of the scar formation is not apparent immediately. The same holds true for Šafář's method. The author has devised an electrode which includes a thermoelectric couple by means of which the amount of heat at the point of application can be measured. He describes a series of experiments done on egg albumin and beef muscle mediums and on rabbit eyes. From a study of the effects of different temperatures ophthalmoscopically and histologically in his experiments on rabbits, Coppez concludes that a temperature of 80 C. is the least harmful and most effective. The use of a pyrometric electrode adds greatly to the security and the constancy of this form of treatment.

S. B. MARLOW.

ETIOLOGY OF HEREDITARY ATROPHY OF THE OPTIC NERVE (LEBER'S DISEASE). L. WEEKERS and R. HUBIN, Arch. d'opht. **50:** 241 (April) 1933.

The observation of a patient with typical Leber's disease whose famliy history could be traced back to the fourteenth century has suggested a theoretical explanation for this disease. In the records of this family no trace of the disease could be discovered up to the time that three brothers married three sisters. Exactly at this time the disease first appeared in all three families; the pedigrees are presented. The observations confirm the already well-known facts concerning the disease. It is sex-limited, but it is not yet possible to explain its occurrence according to the mendelian law. The authors raise the question as to why this disease should appear at a certain time in a family hitherto free from it. They suggest the possibility of the necessity of the union of two unknown predisposing factors which may be present in two separate families. When this union occurs, Leber's disease appears.

S. B. MARLOW.

RESULTS OBTAINED WITH DIFFERENT OPERATIVE TECHNICS FOR DETACHMENT OF THE RETINA BETWEEN OCT. 1, 1930, AND MARCH 1, 1933. F. TERRIEN, P. VEIL and M. A. DOLLFUS, Arch. d'opht. **50:** 331 (May) 1933.

This is a report of the results in one hundred and ten cases in which operation was performed; forty-six of the patients were cured. Of fifty-one patients operated on according to Gonin's method, twenty-five, or 49 per cent, were cured; in the oldest patient complete reattachment remained after two and one-half years. Ten patients were improved, and sixteen obtained no result. In four of the cases classified as cured there were recurrences. Twenty-five patients were treated by suprachoroidal cauterization (Paufique). Twenty-two of these presented tears, eleven, or 50 per cent, being cured, five improved and six unimproved. Of three patients without a tear, one had no improvement, and two had recurrences. The Lindner-Guist operation was performed in twenty cases. Of five patients with tears, one was cured, three improved and one unimproved. Of fifteen patients without tears, five were cured (one recurrence), two improved and eight unimproved. Six patients were treated with multiple puncture and cauterization (Sourdille). Eight were treated by diathermic coagulation (Weve). Four patients without tears obtained no result. Of four with tears, one was cured and three improved. Further study of their observations led the authors to point out that the results correspond to the exactness with which the operation is carried out in relation to the tear. There was perfect localization in 80 per cent of their cases. In the eighty-four patients with tears, cures resulted in 45.5 per cent. In the twenty-six patients without tears, cure followed in only 30 per cent. In the order of effectiveness, the authors list suprachoroidal cauterization (more than 50 per cent cures), thermopuncture (Gonin; 49 per cent cures) and the Lindner-Guist operation (30 per cent success). They have had too little experience with diathermocoagulation to draw positive conclusions, but state that the results are favorable. Complications have been rare in their experience, retinal hemorrhages being the most frequent. Their failures have been due to nonobliteration of the tear. A failure should not be definitely recorded until a sufficient period has elapsed, for sometimes a late reattachment may take place. The authors have used Gonin's method of localization without appreciable error. They believe that Gonin's operation should be used for single, small, easily accessible tears. When the tears are large and multiple, suprachoroidal cauterization should be employed. In spite of the favorable operative results, one must not forget the favorable effect of the injection of mercury oxycyanide subconjunctivally and into Tenon's capsule on recent flat detachments without tears.

S. B. MARLOW.

MY EXPERIENCES WITH ELECTROCOAGULATION IN DETACHMENT OF THE RETINA. J. GONIN, Arch. d'opht. **50:**336 (May) 1933.

Recognizing that thermopuncture is not suitable for all cases of detachment of the retina, Gonin believes that a barrage of adhesions should be produced as suggested by the Guist-Lindner technic or some similar method. The simplest manner of accomplishing this is by means

of electrocoagulation, as advocated by Larson, Weve, Safar and others. He has given this method a trial in thirty cases, and records briefly the records of sixteen of the most instructive cases. He has made use of two methods: (1) external application to the sclera and (2) endothermic puncture. As a result of these personal experiences Gonin believes that the high frequency current is not likely to supplant thermopuncture, but that it can advantageously replace it when it is necessary to create a chain of adhesions to surround either a large tear close to the choroid or a large disinsertion below, or in a large detachment in which it is not possible to determine its origin. It can complete a thermopuncture when this is insufficient to close a large tear. Gonin believes that if he had made his first therapeutic trials with either chemical cauterization or electrocoagulation he would have had difficulty in reaching exact conclusions as to the conditions under which they could or should act to close retinal tears.

S. B. MARLOW.

FACTOR OF ANGIOSPASM IN THE PATHOGENESIS OF RETINITIS PIGMENTOSA, WITH NEW THERAPEUTIC INDICATIONS. M. CORRADO, Ann. di ottal. e clin. ocul. **61:** 43 (Jan.) 1933.

The literature bearing on angiospasm in retinitis pigmentosa is reviewed. The author employed acetylcholine, an active vasodilator, in two typical cases. In one vision improved from 4/10 to 7/10 and from 3/10 to 6/10 immediately after one intramuscular injection of 0.1 Gm. Injections were continued for two months, during which time a slight further increase in central vision (to 8/10) occurred in the right eye, while it remained the same in the left eye. The visual fields showed slight improvement in the right eye only. In a second case, vision improved from 7/10 to 9/10 and from 7/10 to 10/10 following injections. The visual fields also showed definite improvement. Apparently neither case was followed up longer than two months. Corrado advises a trial of the method.

S. R. GIFFORD.

THE CLINICAL IMPORTANCE OF "ATYPICAL" RETINITIS PIGMENTOSA. K. VELHAGEN, JR., Klin. Monatsbl. f. Augenh. **88:** 1 (July) 1932.

The term atypical pigmented retinitis, used by Franceschetti and Best in Schieck's handbook, comprises conditions described as "atrophy of the optic nerve and retina with pigment infiltration," and as tapetoretinal degeneration. The author reports three cases of atypical pigmented retinitis which were complicated by fatty dystrophy, idiocy and polydactylism in one instance.

Two cases belonged to the familial amaurotic idiocy group, described by Spielmeyer and Vogt, and the third to the Laurence-Biedl syndrome. The literature and also the cases show that atypical pigmented retinitis is characteristic for both diseases, although exceptions occur. The question of the relation of ordinary pigmented retinitis to the ocular changes in these two degenerative diseases is still unsettled; clinical considerations, however, suggest a separation. Velhagen's cases and a number of borderline cases of the Laurence-Biedl and the Spielmeyer-Vogt syndromes give rise to the question of the importance of the hypophysis and of the midbrain for the genesis of tapetoretinal degeneration. Proof

could not be established that a primary disease of the hypophysis-midbrain system causes secondary tapetoretinal disease. The clinical value of atypical pigmented retinitis consists in its suggestion that retinocerebral degeneration may be present, the prognosis of which is far more unfavorable than that of ordinary pigmented retinitis.

K. L. STOLL.

THE PROBLEM OF GENUINE DETACHMENT OF THE RETINA. R. DEUTSCHMANN, Klin. Monatsbl. f. Augenh. **88:** 441 (April) 1932.

The author states that the occurrence of detachment of the retina can be explained in a physical way only. He agrees with Baurmann that the difference between subretinal and preretinal pressure is of the utmost importance. An intact retina cannot become detached as long as the preretinal pressure is higher than the subretinal. Intact retinas may become detached by transudation from the choroidal vessels, if the preretinal pressure is the lower one. In the first instance a primary hole, occurring from any cause in the retina, may produce a detachment through escape of liquefied vitreous beneath the retina. Suction or shrinking cords in the vitreous may cause detachment and secondary holes, if the preretinal pressure is lowered. The same may happen in myopic eyes. Deutschmann discusses divers theories of the etiology of the retinal tear. He thinks that it would be more correct to attribute the cause of retinal detachments to pathologic disorder of the lamina elastica. Observations of Behr and Sourdilles suggest that detachment is a symptom of a severe disease of the eye, and that the perforation of the retina is a symptom of the detachment, necessary for its repair. Deutschmann expresses his satisfaction that a number of authors, Schieck, Wessely, zur Nedden and others, recently came to doubt whether a perforation was necessarily the cause of a detachment. The enthusiasm for ignipuncture diminished recently, and warnings against this operation are expressed. More promising than ignipuncture is the attempt to produce closure of the perforations by means of chemicals, for instance, tincture of iodine, or by trephining of the sclera, as practiced by Guist, Lindner and the author. He combined these methods by an incision through the retina next to the scar of the ignipuncture; three of the author's own cases are reported. Deutschmann recommends his method of "refilling" the vitreous as a last resort in old detachments with very low pressure in the vitreous, when other procedures have failed.

K. L. STOLL.

EXPERIENCES WITH THE GUIST-LINDNER OPERATION IN DETACHMENT OF THE RETINA. BARTELS, Klin. Monatsbl. f. Augenh. **89:** 824 (Dec.) 1932.

Bartels performed the Guist-Lindner operation for detachment of the retina nineteen times in fifteen patients. Both eyes of one woman had to be operated on three times for recurrences. No result was obtained in four cases with total detachment and no visible rupture. In seven cases with partial detachment and one or two ruptures, some of them very large, cure was obtained for periods of up to one year. The age seems to be of no consequence, and the visual results were

better for close range than for distance, probably because of opacities of the vitreous. Bartels was surprised to find the peripheral field of vision normal, and in two cases only a small paracentral scotoma after the operation. The cause of the latter phenomenon remained unknown; no changes in the maculae could be observed.

The number of holes made ranged between six and nineteen. Large hemorrhages were observed in three eyes, ptosis occurred in one case as the probable result of extensive severing of the lid and of the cauterization, and double vision was caused by extensive advancement of the internal rectus muscle, which, however, had not been severed. Permanent paresis of the pupillary branch of the oculomotor nerve in one case was explained by retrobulbar lesion during the operation. Bartels prefers Guist's method in ruptures of the ora serrata, although he obtained a good result with Weve's diathermy. Opacities of the lens were not observed. Best results with Guist's method are obtained in partial detachment with a distinct rupture. K. L. STOLL.

Trachoma

UNILATERAL TRACHOMA. D. CATTANEO, Rev. internat. du trachome **9:** 1 (Jan.) 1932.

In a series of 1,600 patients with trachoma, the disease was apparently confined to one eye in 16, but further investigation of these cases with the slit lamp demonstrated that both eyes were in reality involved, the disease process in one eye being so mild as to escape ordinary examination. J. E. LEBENSOHN.

TRANSMISSION OF TRACHOMA TO EUROPEAN CHILDREN IN INFECTED REGIONS. F. MOTAIS, Rev. internat. du trachome **9:** 30 (Jan.) 1932.

Trachomatous infection generally occurs in infancy, and then from contamination by those infected—usually the mother or the nurse. Europeans should be warned that their children, when taken care of by native domestics, are exposed to this danger. J. E. LEBENSOHN.

CONSERVATION OF THE TRACHOMATOUS VIRUS. V. BARONI and D. MICHAÏL, Rev. internat. du trachome **9:** 31 (Jan.) 1932.

Fragments of trachomatous conjunctiva were excised and their infectivity maintained in the following mediums: (1) equal parts of glycerin and Tyrode's solution at 68 C. and (2) human plasma maintained at the temperature of the anterior chamber, 21 C., which had been inactivated by a previous heating to 56 C. for thirty minutes. In testing for infectivity, the material was removed from the medium after an interval of from several hours to three days, and inoculated into the conjunctiva of blind human eyes. An acute diffuse conjunctivitis appeared after from five to nine days, followed in from four to five days by typical follicles of trachoma. Animal mediums (aqueous and plasma from rabbits and cats) constantly rendered the trachoma virus inactive. J. E. LEBENSOHN.

EXPERIMENTAL DATA ON TRACHOMA. D. MICHAIL and P. VANCEA, Rev. internat. du trachome **9:** 33 (Jan.) 1932.

Experimental trachoma has been produced on blind human eyes by Proctor, Morax, Addario, Taboriski and others. The present authors scarified the conjunctiva of the lower culdesac and gently massaged this area with fragments of trachomatous tissue, or with the material obtained from therapeutic expression of trachomatous follicles. In all eight cases, after from five to seven days an acute diffuse conjunctivitis followed; from four to five days later typical granulations appeared, which became increasingly numerous in the succeeding weeks. Experimental trachoma begins acutely, and subsides into a subacute stage, in contrast to clinical trachoma, which is chronic from the start.

Experimental trachoma occurs only on direct contact of the virus with the conjunctival surface. Transpalpebral injection of trachomatous material not only gave regularly negative results but seems to produce some degree of local immunity to subsequent trachomatous infection.

In two cases of so-called monolateral trachoma, though the authors were unable to transmit the disease from the affected eye, they could infect the apparently healthy eye of the patient with the trachomatous virus. They consequently suggest that "monolateral trachoma" is not true trachoma, but only a simulating condition. J. E. LEBENSOHN.

TREATMENT OF TRACHOMA BY CHAULMOOGRA OIL. E. DELANOË, Rev. internat. du trachome **10:** 87, 1933.

In a retort to Pages' criticism, Delanoë presents an extensive array of testimony to the effect that the treatment she sponsors, vigorous massage with chaulmoogra oil, is simple, painless and analgesic, and rapidly ameliorates all stages of trachoma. J. E. LEBENSOHN.

THE MICROBIOLOGIC ETIOLOGY OF TRACHOMA. P. THYGESON, Rev. internat. du trachome **10:** 129 (July) 1933.

A review of the bacteriology of trachoma was given at the Fourteenth International Ophthalmological Congress at Madrid. Because of the fact that the author personally has recovered the bacillus of Noguchi (Bacillus granulosis) from cases in various parts of the world, he believes firmly in its etiologic nature. Because it took so many years to discover this organism, it is readily seen why a filtrable virus might be thought to be the cause. L. L. MAYER.

RESULTS IN ONE HUNDRED AND FIFTY INTRADERMAL REACTIONS OF TRICOIRE IN TRACHOMA. J. SEDAN, Rev. internat. du trachome **10:** 145 (July) 1933.

In this report to the First Mediterranean Congress of Hygiene, the author reviews the Tricoire reaction and results previously reported. The following conclusions concerning 150 persons with the florid type of trachoma are made:

1. In nontrachomatous subjects the reaction is negative in the majority of cases.

2. Of the 150 patients examined, the reaction was (*a*) positive in 92, or 61.33 per cent; (*b*) doubtful in 24, or 16 per cent, and (*c*) negative in 34, or 22.67 per cent.

Thus, three-fourths gave a positive reaction. The author makes a plea for the wider use of this test.

L. L. MAYER.

LOCAL IMMUNOTHERAPY IN TRACHOMA. M. ESTEBAN, Arch. de oftal. hispano-am. **33:** 91 (Feb.) 1933.

This investigation is based on Besredka's studies of local immunity. The author refers to the forgotten vaccine therapy of Pietro Vancea and to the observations of Nicolle and Cuenod, to which he objects on the basis of the unnecessary production of antibodies in the blood when only a local action is required.

He obtains his material by bloodless squeezing of trachoma granulations with artery forceps and by superficial scraping with a blunt curet. He mixes it with from 5 to 10 cc. of physiologic solution of sodium chloride which is inactivated by heat for one-half hour (60 C.). The solution is dropped on the everted tarsus daily for three or four weeks. Once a week the tarsus is rubbed with a dry gauze pellet until bleeding is induced to promote absorption of vaccine. So far the author has used this material only in the patient from whom it was obtained.

The results in forty-nine previously untreated patients, mostly children, are given in tabular form. These show: cures in 80.95 per cent, improvement in 16.66 per cent and reproduction in 2.38 per cent.

C. E. FINLAY.

Tumors

OCULAR PAPILLOMATA. J. A. DOHERTY, Am. J. Ophth. **15:** 1016 (Nov.) 1932.

Two cases are reported. The first was in a man, aged 56. The left eye was affected. The tumor consisted of a number of mushroom-shaped globules having the appearance of granulation tissue, attached to the sclera by a pedicle. A biopsy showed thickened squamous epithelial cells thrown up into irregular folds by projection of the stroma. The basement membrane of the epithelium was well preserved. The patient died of advanced carcinoma of the stomach before the ocular growth could be removed.

The second case occurred in a woman aged 30. About six months before, a growth had been removed from the inner canthus of the right eye. There were three separate tumors: One was on the edge of the upper lid, one occurred near the edge of the semilunar fold and one, a lobulated growth, was attached to the caruncle. All were removed and radium treatment was given. There has been no recurrence. The histologic changes were similar to those in the growth in the first case. These growths are capable of malignant degeneration.

The literature is reviewed, and the clinical and pathologic characteristics are described.

W. ZENTMAYER.

TUMORS OF THE SEMILUNAR FOLD. U. LUMBROSO, Rassegna ital. d'ottal. **2**: 93 (Jan.-Feb.) 1933.

A soft, lobulated, pinkish tumor of the semilunar fold was seen in a woman, aged 42. It proved to be a collection of unpigmented cells resembling those of a nevus. In sections an apparent connection was seen between the overlying epithelium and the characteristic tumor cells. Lumbroso believes that the nevus cells originated from the basal epithelium.

S. R. GIFFORD.

A CASE OF FIBROMA GLOBOSUM OF THE CORNEA. A. MUSIAL, Ztschr. f. Augenh. **80**: 147 (April) 1933.

A child 1 year of age exhibited a half-round, hard, bluish-white mass on the cornea, which prevented closure of the lids. A small cloudy spot had been noticed on the eye of the child when 2 months of age by its mother, who stated that this had gradually increased in size. The surface of the mass was dry, smooth and covered with thickened epithelium. Its appearance suggested a staphyloma. Microscopic examination of a wedge-shaped piece excised showed the mass to be made up of dense connective tissue having spindle-shaped cells with long, thin processes. A few blood vessels were present, and there were areas of hyaline degeneration. The tumor was covered with several layers of flattened epithelium. Although the few cases of this growth already described have been attributed in origin to corneal scars, the author suggests that in this case the mass probably resulted from a developmental disturbance dating back to the third or fourth month of the embryo at the time that the primary optic vesicle undergoes differentiation into opaque scleral and transparent corneal tissue.

H. L. BEGLE.

Uvea

HETEROCHROMIA OF THE IRIS. WANG LAN-TING, Chinese M. J. **18**: 963 (Oct.) 1932.

A case of heterochromia of the iris is reported in a 21 year old Chinese, which is considered a true case of Fuch's heterochromia and the first one to be reported among the Chinese, in whom the diagnosis can be made only with the greatest difficulty on account of the brown irides of the entire population. Clinical examination of the right eye showed the characteristic bedewing on the posterior surface of the cornea, together with about twenty white precipitates evenly distributed. The iris in the right eye was brown, but slightly lighter than that in the left eye, and had a uniform spongelike structure, not only in the pupillary, but in the ciliary, zone. The lens showed an intumescent cataract, with water-clefts around the anterior senile suture system. The vision was reduced to perception of hand movements at 60 cm.

S. H. MCKEE.

Visual Tracts and Fields

FIELDS OF VISION IN CONNECTION WITH INTRACRANIAL LESIONS. ADAMS A. MCCONNELL, Brit. M. J. **2**: 226 (Aug. 5) 1933.

The author summarizes the visual pathways briefly as follows: Fibers from the upper half of the retina lie dorsal from the eye to the cortex; those from the lower half lie ventral, while the macular fibers lie between.

The work of Poliak, Holmes, and others indicates a fixed geometric projection of the retinas on the striate cortex. No segment, not even the fovea centralis, has a diffuse or bilateral cortical representation.

In the right striate area, fibers from the most nasal part of the left retina (unpaired field) terminate in the extreme anterior end of the fissure; those from the right halves of the retinas terminate more posteriorly, and the most posterior portion receives the fibers from both homonymous hemimaculae. Or, in terms of fields, the peripheral part of the left visual field is projected on the anterior part of the right visual cortex, and the central part on the posterior part. The floor of the fissure represents the horizontal meridian of the retina.

The author divides the temporal lobe into a pregeniculate and a postgeniculate area by a coronal plane at the anterior portion of the external geniculate body. This plane divides the temporal lobe almost exactly into halves. He thinks that if a definite "tract" defect of the visual fields can be established, in contradistinction to a "radiation" defect, each part of the temporal lobe has its own burglar alarm. He describes seven cases of tumors involving the temporal lobe in which the diagnosis was made, not of a tumor of the temporal lobe, but of a tumor involving the visual radiation, and operation planned accordingly.

W. F. DUGGAN.

Vitreous

THE CHEMISTRY OF THE VITREOUS. WINTERNITZ and STARY, Arch. f. Augenh. **107**: 191, 1933.

Although the aqueous, the vitreous and the spinal fluid are somewhat similar in chemical composition, i. e., protein content, high chlorides and lowered calcium, there are a number of differences which make it advisable to analyze these fluids more carefully. Only by such a comparison can one say whether the fluids are formed from blood by the same mechanism.

The present studies concern determinations of calcium and magnesium in the aqueous and vitreous of horses. The eyes were enucleated, and aqueous and vitreous from the same eye were analyzed. In some of the experiments reported it is not made clear whether or not the blood was withdrawn from the same animal.

The calcium content of the vitreous was found to average 6.4 mg. per hundred cubic centimeters, whereas the calcium content of the blood serum averaged 12.1 mg. The calcium content of the aqueous averaged 7 mg. per hundred cubic centimeters. The calcium content of the vitreous thus agrees with the conception of the vitreous as a dialysate. The author then dialyzed blood serum through an inert semipermeable membrane against vitreous fluid, and at the end of forty-eight hours

found no change in the concentration of the calcium content of the vitreous. The same difference which existed before dialysis persisted afterward. This is confirmatory evidence that the membrane separating the blood from the vitreous is an inert semipermeable membrane.

The magnesium content of the vitreous averaged 1.92 mg. per hundred cubic centimeters. That of the blood serum averaged 1.57 mg., and that of the aqueous, 1.33 mg. The magnesium content of the vitreous is higher, therefore, than that of the blood serum and does not correspond to the portion of the blood magnesium which is known to be free and diffusible.

The author dialyzed blood serum against vitreous fluid and found at the end of the period a complete change in the disposition of the magnesium in the two fluids. Before dialysis there was more magnesium in the vitreous, whereas after dialysis there was more magnesium in the blood serum.

The vitreous fluid differs from the aqueous, therefore, in its chemical composition, and it is not as comparable to a dialysate as is the aqueous. If dialysis is the mechanism concerned in the formation of vitreous, there must be other mechanisms present which completely alter the character of the fluid so that it can no longer be recognized as a dialysate.

F. H. ADLER.

BANDLIKE FORMATIONS IN THE VITREOUS: REPORT OF TWO CASES. G. KLOESS, Klin. Monatsbl. f. Augenh. **88**: 161 (Feb.) 1932.

The first chapter of this paper is devoted to the etiology of bands in the vitreous. Giving due consideration to the three main theories of the origin of the vitreous, the author expresses his opinion that the bands in the vitreous are remnants of the embryonic vascular system, which would support the mesodermal theory. Kloess also considers the differential diagnosis and the types of malformation, inhibition of normal development or sequelae of pathologic processes that may intervene.

He reports two of his own cases. The first patient, a boy, aged 11 years, sustained an injury of the right eye sufficient to cause a tear of a congenital circumpapillary and epipapillary membrane, several strands of which extended into the vitreous. A hemorrhage, resulting from the injury, was supposed to have been caused by the rupture of blood vessels contained in the strands. Two months later the opacities of the vitreous had absorbed.

In the second case, that of a woman, aged 30, the left eye showed an epipapillary membrane and strands in the vitreous. These were remnants of Cloquet's canal and of the hyaloid vessels. An inflammatory origin was excluded.

K. L. STOLL.

BILATERAL, SPONTANEOUS RING-SHAPED OPACITY OF THE VITREOUS (VOGT'S SENILE RING IN THE VITREOUS). ISAKOWITZ, Klin. Monatsbl. f. Augenh. **88**: 369 (March) 1932.

The author refers to a statement of Vogt, who suggested the term "senile vitreous ring" for a small, annular, entoptic formation, which he classed among the degenerative processes of the aging eye (*Klin.*

Monatsbl. f. Augenh. **75:** 463, 1925). Isakowitz reports the case of a man, aged 58, in whom he observed the development of an annular opacity in the vitreous, located symmetrically in the two eyes. The patient noticed a shadow in his right eye for nine months and in his left for three months, about 8 degrees from the point of fixation in each eye. The centers of the rings were clear and permitted a distinct view of the retina and central vessels. The ring in the right eye had a veil-like projection; that in the left was torn and slightly twisted, bearing a fine veil at the tear. The rings pendulated about 3 mm. in front of the retina; slight degenerative changes of the vitreous were noticed with the slit lamp. Both eyes were emmetropic; vision was normal. The rings were of systemic origin, in the author's opinion. He was uncertain, however, whether they were derived from Cloquet's canal or were caused by a ring-shaped dehiscence of the vitreous from the disk.

K. L. Stoll.

Therapeutics

The Treatment of Painful Absolute Glaucoma and Other Eye Diseases with Retrobulbar Injections of Alcohol. J. Fejer, Am. J. Ophth. **15:** 135 (Feb.) 1932.

The author uses the method of Grüter, who first cocainizes the eye and then injects 1 cc. of 2 per cent procaine hydrochloride to the right and left of the eyeball near the posterior pole by means of a curved Siegrist needle. After five minutes a similar amount of 80 per cent alcohol is injected in the same manner and into the same region. Fifteen minutes later the eyeball is entirely anesthetic, and all the ocular muscles are paralyzed. Inflammation of the orbital tissues follows with protrusion of the globe, chemosis and stain. This injection was used in five cases of absolute painful glaucoma. In the first two, the patients, a woman of 59 and a man of 75, received permanent relief from pain. In the third case the pain was relieved, but the eye had to be enucleated six weeks later because the pain returned. The author attributes this recurrence to the fact that the inner surface of the eyeball was lined with a thin shell of bone. In the fourth case, that of a sailor, aged 63, pain was permanently relieved, but the eye remained hard. In the fifth case pain returned a week after the injection of alcohol. The eye was enucleated and found to contain a large fusocellular melanosarcoma. The author does not recommend this therapy in seeing eyes.

W. S. Reese.

Biologic Treatment of Ocular Herpes. A. N. Martin and E. Diaz Caneja, Arch. de oftal. hispano-am. **33:** 367 (June) 1933.

Five cases of corneal herpes (dendritic keratitis) successfully treated by desensitization with Levaditi's herpeto-encephalitic virus are reported. The treatment is based on modern investigations of the filtrable virus to which herpes is attributed and on the theory of its identity with the virus of epidemic encephalitis confirmed by researches of Blanc and Levaditi.

C. E. Finlay.

LOCALIZED DILATION OF THE PUPIL PRODUCED BY IONTOPHORESIS WITH EPINEPHRINE. G. and A. ERLANGER, Klin. Monatsbl. f. Augenh. **88:** 86 (Jan.) 1932.

The authors obtained protracted mydriasis in rabbits by iontophoresis with epinephrine, using a galvanic current of 2 ma. for two minutes. Epinephrine hydrochloride (dilution 1 : 1,000) was sent from the positive pole through the cornea in a glass tube armed with a zinc rod. The pupil dilated on the side of the electrode if the electrode, prepared with epinephrine, was placed on the sclera and iontophoresis of 2 ma. was used for two minutes. In human eyes similar results were obtained after introducing physiologic solution of sodium chloride and from 3 to 5 drops of a 1 : 1,000 solution of epinephrine under iontophoresis of 1 ma. for one minute. Mydriasis began after from ten to fifteen minutes and lasted for from sixty to a hundred minutes. A solution of 0.25 per cent of calcium chloride, introduced from the positive pole and followed by the instillation of 1 drop of epinephrine about fifteen minutes after the iontophoresis, will produce oval distortion of the pupil toward the electrode. A long period of mydriasis may be produced even with a 1 : 10,000 solution of epinephrine.

Localized mydriasis may be used in an impacted iris, by putting the electrode on the impaction and also diametrically opposite it. This method can be used to good effect in upward displacement of the iris after intracapsular extraction. Prolapses of the iris, seen frequently in dogs, retract promptly.

K. L. STOLL.

TREPHINING OF THE CORNEA IN SERPIGINOUS ULCER AND OTHER DISEASES. R. and G. SONDERMANN, Klin. Monatsbl. f. Augenh. **88:** 189 (Feb.) 1932.

Referring to their previous publication on "the importance of the intra-ocular tension for the healthy and diseased eye" (*Klin. Monatsbl. f. Augenh.* **87:** 450 [Oct.] 1931; abstr., ARCH. OPHTH. **8:** 910 [Dec.] 1932), the authors report their experiences with intra-ocular decompression in serpiginous ulcer. They trephined the cornea in seventy-five cases of this type of ulcer, with an unfavorable outcome in only four eyes. The secondary increase of tension that developed in nine cases could be eliminated by means of an iridectomy or cyclodialysis. The adverse results occurred in persons, aged from 65 to 75, who presented ocular hypertension, so that the effect of the corneal trephining was frustrated by too swift closure of the opening.

The same experience occurred occasionally when a trephine 1 mm. in diameter had been used, wherefore the authors chose trephines 1.5 mm. in diameter. Aside from the intra-ocular tension, the location and size of the ulcer, the physical condition of the eye and the rate of progress of the ulcer are deciding factors in the operation. The higher the age, the less favorable is the prognosis. The virulence of the organisms may be disregarded, and delay should be avoided. Injury to the anterior capsule of the lens can be avoided by adapting the trephine on the cornea in an oblique manner. The "button" or trephining disk of the cornea, including Descemet's membrane, must be removed.

Trephining the cornea is contraindicated in acute tuberculous processes. Good results were obtained in chronic diseases of the uvea and retina, and in types of corneal ulcers other than the serpiginous type. Preliminary paracentesis of the anterior chamber had shown the beneficial effect of lowering the intra-ocular tension in these cases. Only in nine eyes with serous iridocyclitis was the trephining of the cornea performed successfully after careful selection of the cases. The operation may prove beneficial also in other diseases of the eye than those mentioned.

K. L. STOLL.

SURGERY OF RETINAL DETACHMENT WITH PERFORATION IN THE MACULA: REPORT OF CASES. K. LINDNER, Klin. Monatsbl. f. Augenh. **88:** 544 (April) 1932.

Lindner refers to the method of perforating cauterization of tears in the macula, which he used in connection with Gonin's operation. Two methods were employed by the author for the caustic method: 1. Guist, who performs a modified Krönlein operation to bare the posterior pole of the eye, trephines in the region of the macula, and uses the caustic. This method is difficult; it requires several hours for its performance and may cause lesions of the posterior ciliary nerves. 2. Lindner makes an incision through the sclera, as far back as possible; the choroid is not severed. Then he passes a spatula between the sclera and the choroid into the area of the tear. Into this undermined subchoroidal space about 0.02 cc. of a 6 per cent solution of potassium hydroxide is injected through a flat cannula with a lateral opening. The two first cases in which operations were carried out according to this method were demonstrated. The first patient showed an extensive central choroidal patch with retinal hemorrhages. The detachment had disappeared, although a second tear in the lower portion of the retina had not been treated. In the second patient, the tear in the macula and another, nasally situated and above it, were closed, and complete reattachment was obtained. The vision was considerably improved, and the field of vision returned to normal. The possibility of a perforation into the vitreous chamber must be considered. Experiments on animals showed that the potassium hydroxide solution yields the best results of all chemical combinations used.

K. L. STOLL.

RESULT OF TREATMENT WITH ROENTGEN AND GRENZ RAYS IN PARENCHYMATOUS KERATITIS: REPORT OF CASES. F. GNAD, Klin. Monatsbl. f. Augenh. **89:** 446 (Oct.) 1932.

Publications by a number of authors show that belated lesions of the lens may occur even after application of small doses of roentgen rays. Rohrschneider mentioned fifty publications on lesions of this kind up to 1930.

Referring to satisfactory results obtained in a large number of cases of syphilitic parenchymatous keratitis and published by more than a dozen authors, Gnad reports the observations made at Elschnig's clinic in Prague.

Comparing the condition in thirty-seven patients treated with roentgen rays with that in patients not subjected to this therapy, Gnad stated that no decided benefit was observed. It is not justifiable, in his opinion, to risk producing lesions of the lens, although the course of the disease may be shortened by roentgen therapy in some acute cases. Syphilitic processes should not be treated by roentgen rays, least of all parenchymatous keratitis, the main symptoms of which are due to degenerative changes, not to primary inflammation. Four patients with typical parenchymatous keratitis showed negative Wassermann reactions but strongly positive tuberculin reactions. All of them were treated with tuberculin, and all but one with roentgen rays, with satisfactory results in each case. Roentgen therapy seemed to be indicated in this type of case.

No advantage in the use of the Bucky tube was recorded in four patients with parenchymatous keratitis with positive Wassermann reactions of the blood. Lesions of the deeper layers of the eye are not likely to occur, as this tube has small penetrating power.

K. L. STOLL.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Jan. 19, 1933

LEIGHTON F. APPLEMAN, M.D., *Chairman*

PLASTIC OPERATION BY PEDICLE FLAP. DR. CHARLES R. HEED and DR. GEORGE CROSS.

Following a severe explosion, this patient, a miner, suffered extensive burns and lacerations of the face, with ruptures of both eyes. Repairs of the alae of the nose and of the angle of the mouth were carried out by Dr. Warren B. Davis. Later, correction for ectropion of the left lower lid was performed by Dr. Cross by means of a pedicle flap taken from the right frontal region.

DISCUSSION

DR. CROSS: This case presented unusual difficulties. The deformity and scarring were so great that we could not use the skin from the left side of the face to form grafts: We decided to utilize the skin from the right frontal region to correct the ectropion of the left lower lid, the blood supply coming from the area at the base of the nose.

This preliminary operation was performed about twelve days ago. After allowing a month for the circulation in the graft to become thoroughly established, we shall open the lower incision and remove the excess tissue and restore the bridge of the nose to its original condition.

It is interesting to see the large amount of tissue that one can remove from the forehead and yet produce practically no deformity. To prevent a wide, disfiguring scar, it is essential that the fascia be sutured securely when the incision is closed. This prevents stretching and dimpling. The incision surfaces were brought together with mattress sutures of silkworm gut, and the edges of the skin with fine black silk sutures.

ERYTHEMA EXUDATIVUM MULTIFORME. DR. EDWARD A. SHUMWAY.

This case of erythema exudativum multiforme involves the mucous membranes of the eyes, nose and throat and vaginal tract, with minor lesions of the skin of the forearms. In all probability, the condition was caused by long use of liquid petrolatum, which produced an avitaminosis in a patient who was also susceptible to the toxic effect of phenolphthalein.

Dr. Shumway refers to another patient seen by him in consultation who presented the same symptoms and had a similar history.

DISCUSSION

DR. F. D. WEIDMAN: From the dermatologic standpoint, I am unable at this moment to make a diagnosis either of erythema exudativum multiforme or of an eruption due to phenolphthalein. The lesions are regressing, but there is no residual pigmentation as in a phenolphthalein eruption. I do not know whether therapeutic tests have been applied, that is, whether another dose of phenolphthalein has been given to see whether or not the eruption will reappear. I feel that the lesions are more likely to prove to be due to an avitaminosis. Lesions are present on the gums, but they are scarcely suggestive of scurvy.

I have only one constructive suggestion to make—that the examination of the lesions, especially in the mouth, include a search for fungi, notably Monilia.

DR. S. S. GREENBAUM: There are many causes of erythema multiforme, but, in this case, I believe that the eruption is due to a drug, phenolphthalein. I did not have avitaminosis in mind, and I do not believe that such a condition caused this eruption. Only one avitaminosis that I know of involves both the eyes and the mouth, and that is xerosis or keratomalacia, a condition in which there is a shrunken, dried state of the eyes as well as involvement of the mouth (atrophy of the lacrimal and salivary glands). The only avitaminoses known to involve the mouth are scurvy, sprue, pernicious anemia and pellagra.

DR. MAXWELL HERMAN: I saw this patient in August, 1932, and at that time she presented typical catarrhal conjunctivitis with a flocculent exudate; the particular feature was that the secretion was rather solid and fibrinous and almost like pseudomembrane.

My diagnosis was some form of allergic reaction, similar to vernal conjunctivitis, which would probably clear up with the advent of cold weather. Not until the end of October did the patient manifest any involvement of other mucous membranes. This first appeared on the cheeks and the gums. The point that I want to stress is that the condition started in the conjunctivae without involvement of other mucous membranes.

DR. H. C. WOOD, JR.: After listening to the account of the case, I feel certain that if it is a case of avitaminosis, there must be some other contributing factor present. I have never heard of any avitaminosis that could give such a combination of symptoms and lesions as this patient presents. I lean strongly toward the opinion that phenolphthalein had something to do with it. While there are no cases on record, so far as I know, of phenolphthalein giving rise to lesions involving the eye, there are numerous cases of cutaneous lesions of this type due to phenolphthalein and a few cases of involvement of the mucous membranes. If one could suppose a second cause for the lesions of the eye, all the other symptoms, I think, might be explained as the effect of phenolphthalein.

So far as I have read, most of the cases of poisoning from phenolphthalein have been caused by large doses, but in abnormally sensitive persons moderate doses can do harm. I have not heard mentioned the dose used in this case, but I think it is highly plausible that the phenolphthalein caused the cutaneous lesions, at least.

DR. EDWARD S. SHUMWAY: My associates and I hesitated to give experimental doses of phenolphthalein, as cases have been reported

which progressed to complete destruction of the cornea and loss of vision, and it did not seem wise to give more phenolphthalein in this case. We did not make a cystoscopic examination, as local conditions would have made it very painful.

The urine and feces were tested for the presence of phenolphthalein, but none was found; this was to be expected because the patient had not taken any of the drug for five or six weeks.

I should be glad to allow Dr. Weidman to take some of the secretion and make a culture of it on his special medium in order to help us in making a diagnosis.

THE USE OF EPINEPHRINE IN OPHTHALMOLOGY. DR. H. MAXWELL LANGDON.

In 1911, Rollet and Curtil reported that subconjunctival injections of epinephrine produced a marked fall of intra-ocular tension.

In 1913, Erdman reported that subconjunctival injections of epinephrine produced dilation of the pupil and led to breaking of synechiae that had not been broken by mydriatics.

In 1920, Shahan and Post reported that cocaine and epinephrine caused a drop in ocular tension, followed by a rapid rise.

In 1921, Knapp reported the action of epinephrine on the pupil in sixty-five cases of primary glaucoma. The drug was administered entirely by instillation, and the results were variable.

In 1922, Fromaget reported a case in which the injection of 3 cc. of procaine hydrochloride and epinephrine for anesthetization before operation for glaucoma produced disappearance of glaucomatous symptoms.

In November, 1923, Hamburger reported before the Berlin Medical Society that subconjunctival injections of epinephrine produced dilation of the pupil and a marked reduction of tension in glaucomatous eyes. One month later Fehr reported sixteen cases in which the eyes had been treated in this way.

In 1924, Gradle reported some of his work begun in 1912, confirming the findings of the other observers.

In 1920, Hamburger reported his work on the use of dextrorotatory epinephrine instead of the more common levorotatory form. He found that he obtained the best results when he used a combination of these. He claimed that this combination did not produce alteration in the general blood pressure. Four cases are reported by Dr. Langdon which illustrate the effects of epinephrine on various manifestations of increased ocular tension. In these cases, the epinephrine was used on pledgets of cotton placed beneath the closed lids and kept there from twenty to twenty-five minutes.

DISCUSSION

DR. C. E. G. SHANNON: Dr. Magitot of Paris suggested that 4 minims (0.24 cc.) of a 1:1,000 solution of epinephrine hydrochloride be injected subconjunctivally about fifteen minutes prior to extraction of a cataract in order to secure full dilation of the pupil. The dilation is maintained throughout the operation. In addition, I have found this use of epinephrine helpful in preventing hemorrhage into the anterior chamber, which is most annoying at times.

COLOBOMA OF THE OPTIC NERVE. DR. JAMES S. SHIPMAN (by invitation).

This article will appear in full in a later issue of the ARCHIVES.

OCULAR LESIONS CAUSED BY SINUSITIS. DR. NATHAN B. STAUFFER (by invitation).

Some patients have blurred vision without opacities in the media; others complain of intense itching of the conjunctiva or, more frequently, of headaches without visual or refractive changes; others are troubled with swelling of the lids or epiphora. In some cases, moreover, the ophthalmologist finds retinal changes and desires a report as to the nasal condition. Fourteen cases are presented which illustrate ocular complications in sinusitis.

The question naturally arises: How does the sinus disease affect the eye—by disturbing the blood supply (that is, by pressure), by continuity, by infection of the blood stream, by way of the lymph channels or by a systemic toxemia?

DISCUSSION

DR. H. MAXWELL LANGDON: In my experience it is rather unusual to find a purulent secretion in the sinuses. The report which one ordinarily gets is that the rhinologist is unable to find enough pus present to warrant an operation or make him reasonably sure that the ocular condition is due to the sinus disease. It seems to me that itching of the eyes is an unusual symptom of sinus disease; when such a condition is present, one thinks far more frequently of vernal catarrh or of hyperacidity. In considering the involvement of the sinuses in Dr. Stauffer's cases, I note that the frontal sinuses were involved in 78 per cent of the cases, the ethmoid in 58 per cent, the maxillary in 38 per cent and the sphenoid in 7.8 per cent. Of the sinuses which produce ocular involvement, it seems to me that the ethmoid and the sphenoid do most frequently, and the maxillary, the next most frequently. The frontal sinus seems the least likely to cause ocular complications.

DR. CHARLES R. HEED: In regard to diplopia, I must confess that I give no treatment for it. The patients usually recover. This is especially true in diseases of the posterior ethmoid sinuses.

DR. T. B. HOLLOWAY: I hoped that some definite statement would be made in regard to retrobulbar neuritis and that Dr. Stauffer would express his opinion concerning the relation between that condition and the accessory sinuses.

DR. NATHAN B. STAUFFER: In answer to Dr. Langdon's statement that pus is not found so often in his cases as in mine, I think that this is explained by the fact that the disease was advanced further in my cases than in his.

In reply to Dr. Heed, who spoke of patients with diplopia getting well without treatment, I have found that this is more often true if the posterior ethmoid sinuses are affected.

Dr. Holloway wants to know about retrobulbar neuritis. I think it must be due to inflammation or to pressure.

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(Continued from page 377)

MEDICAL TREATMENT OF RETINAL DETACHMENT. DR. G. OVIO, Rome, Italy.

Even before there was any definite understanding of the nature of retinal detachment and when it was classed, together with a large number of other amauroses, as a general disturbance of vision, various forms of medication were advised. Surgical procedures followed at a comparatively early date, thanks to Ware's study (1804) advising scleral puncture. This form of treatment, at first rather indefinite, was crystallized, so to speak, when Samelsohn (1875) suggested the pressure bandage and continuous rest in bed with the patient lying flat on his back. Of the various procedures and medicinal therapeutic measures since added to these two, the principal ones are keeping the patient in a dark room, diaphoresis, injections (instillation?) of physostigmine or atropine, electrolysis, massage, the administration of iodides or other drugs (mercurials, epinephrine, tuberculin), a salt-free diet, subconjunctival injections and injections into the vitreous. Subconjunctival injections for retinal detachment, introduced by K. Grossmann (1883), won a definite place in practical treatment only after Mellinger's experimental studies (1894) and appeared to mark a definite step in advance in dealing with this serious disease. All the therapeutic procedures mentioned are based on rational, logical conceptions of mechanical cause and effect. For more than half a century there has persisted a recurring alternation between discouragement and enthusiasm. The statistics collected by Dr. Ovio show an average of improvement or recovery in about 22 per cent of the cases.

OPERATIVE TREATMENT OF RETINAL DETACHMENT. DR. A. VOGT, Zurich, Switzerland.

Rest is a factor which must enter into the operative management. Closure of the retinal tear is of the first importance. Many details have to be added to its history. Coccius (1853) was the first to see a retinal tear; then von Graefe, Hansen and Schweigger. De Wecker (1870) recognized that the tear was the cause of detachment. Leber (1882) expressed the general dictum that every ophthalmoscopically diagnosable (acute) detachment of the retina originates in a retinal tear. Ignipuncture was introduced by de Wecker, Martin and de Luca (1881). Schoeler (1889) first attacked directly the tear and the original starting point of the detachment with tincture of iodine. Deutschmann (1896) was the first to puncture the tear itself. Galezowsky first used ignipuncture systematically on the tear and also sucked up the retroretinal

fluid. Gonin carried the idea to a triumphant conclusion. Without his work the present-day successes could not be thought of. The formation of a tear is either traumatic or spontaneous. In the latter case there are tissue changes leading to destruction of the retina and vitreous. More and more ophthalmologists are becoming settled in the conviction that spontaneous detachments are due to retinal tears, that de Wecker and Leber were right, and also that lowered intra-ocular tension is a direct result of the tear. Accurate localization is of prime importance. Both Vogt and Gonin prefer the ophthalmoscope to the various localizing apparatus for this purpose. A recapitulation of present-day methods is given. The special task for the future will be to lay down precise indications for the various procedures. Possibly needle-diathermy deserves the preference. If the electrocautery is used the burning should last only from one to two seconds and should be carried out with a needle, not with a loop. Vogt works with the Weve needle in cases in which the tear lies in close apposition to the choroid, and exclusively with the galvanocautery in the case of extensive tears, such as disinsertions at the ora serrata. Details of these procedures and of others which do not aim at closing the tear are given.

DISCUSSION

Dr. Diaz-Caneja, Valencia, Spain: The photographs which I am presenting show the various types of retinal fold formation and their differentiation in various forms of retinal disease. The ophthalmoscopic camera is of great practical diagnostic value.

Results of Experimental Detachment of the Retina. Dr. C. Dejean, Montpellier, France.

On dissecting the enucleated animal eye one finds that after a scleral incision the pigment epithelium, choroid and sclera separate together very easily from the retina. The vitreous detaches easily from the retina, if the latter is held firmly in place. Occasionally, it is true, small portions of the retina stick to the vitreous so that minute tears develop. In the living animal eye it was generally not feasible to bring about retinal detachment by means of small incisions. But if a large amount of vitreous was allowed to escape through an extensive breach the retina became folded and detached in consequence of the hypotony of the globe. Hence, marked loss of tension and collapse of the globe are the most dependable factors in the experimental production of detachment. They may be of practical importance in the clinical variety, as well.

Physical Chemistry of the Subretinal Fluid in Myopic Detachment. Dr. M. Jasinski, Poland.

Dr. Jasinski reports tests and analyses of the transparent, light yellow, fibrin-free fluid drawn off with the hypodermic needle. Tests with the refractometer showed marked variation in the albumin content. The osmotic pressure, measured with Barger's micrometer, is lower than that of the aqueous. The electric resistance was measured by Kohlrausch's method. The resistance of the serum is higher, and that

of the aqueous lower, than that of the subretinal fluid. The viscosity, determined by Hess' method, is very variable, as is the relation between albumin and globulin. Cellular analysis and cytology showed only a few white blood cells. The Umber test was negative. To sum up, the subretinal fluid is generally not of an inflammatory nature.

RESEARCH ON THE ETIOLOGY OF IDIOPATHIC RETINAL DETACHMENT. DR. D. SABBADINI, Rome, Italy.

In one hundred and thirty cases careful search was made for a general predisposing cause. In most of the cases (thirty-five) tuberculosis played the principal etiologic rôle; in others (thirty), syphilis, and in the rest, arteriosclerosis, cardiorenal disease or diabetes. The cases of tuberculosis were generally instances of pulmonary involvement which although not easily demonstrated clinically were definitely localized by means of teleroentgenography. According to Dr. Sabbadini, the ocular localization which leads to detachment is apparently a metastatic deposit (beginning of Ranke's secondary period); such metastases may represent the one and only organic focus derived from the primary seat of the disease (Ranke's tertiary period). General treatment before, during and after surgical intervention is a decided aid to recovery. The so-called myopic changes which accompany idiopathic detachments in near-sighted eyes are nothing else than uveal manifestations of tuberculous or syphilitic foci, particularly in juvenile myopes.

ETIOLOGY OF DETACHMENT OF THE RETINA. DR. J. LOPEZ ABADIA, Bilbao, Spain.

One may divide the cases into two categories, the pathologic and the traumatic; the latter group, again, into cases due to concussion and those developing as a result of injury by blunt violence. The mechanism of retinal detachment was fully discussed as early as 1906 by Dufour and Gonin (in Lagrange and Valude: Encyclopédie française d'ophtalmologie, Paris, O. Doin, 1903-1910). The formation of a tear and the gradual ingress of vitreous are significant only in exceptional cases.

RETINAL DETACHMENT AND VASOMOTOR NEUROSIS. DR. HORNIKER, Trieste, Austria.

Dr. Horniker examined seventeen patients with idiopathic detachment for signs of vascular neurosis (punctate opacities of the lens and degenerative changes in the endothelium of Descemet's membrane [*Arch. f. Ophth.* **119:** 448, 1928]). The signs were positive in all of these patients and in a series subsequently studied. Clinical findings as well as the results of microscopic-capillary tests confirmed the diagnosis of vasomotor neurosis. The blood pressure in the brachial artery of these subjects was compared with that of patients with glaucoma. These, as is well known, show marked hypertension with extreme oscillation of values and a decided difference between the two sides. In retinal detachment, the arterial pressure is comparatively low, oscillations are slight, and the difference on the two sides is inconsiderable. The condition is probably one variety of the protean manifestations of

vasomotor neurosis, caused by increased permeability of the capillaries. Functional anomalies of the capillaries may be the basis for the foci of peripheral cystic degeneration which are now looked on as the probable starting point of many, if not all, tears.

SPONTANEOUS CURE OF BILATERAL RETINAL DETACHMENT IN THE COURSE OF BRIGHT'S NEURORETINITIS. DR. E. HARTMANN, Paris, France.

Broad detachment in the lower half of the retina occurred in a case of severe nephritis; reattachment occurred spontaneously.

MECHANICS OF SPONTANEOUS REATTACHMENT OF THE RETINA. DR. C. PASCHEFF, Sofia, Bulgaria.

Nature's cure, as shown by Dr. Pascheff in clinical cases, is accomplished (1) by resorption or (2) by scar formation. One must come to the assistance of these natural processes by bringing about the resorption of the subretinal fluid or the cicatrization of the tear. The first procedure is indicated in cases of detachment without tear; the second, in cases with tear. For the former, treatment by means of osmosis plays an important part; in the second, thermopuncture or diathermopuncture is the therapeutic factor *par excellence,* and closing of the tear, the main point. Finally, detachment is never to be considered as cured unless the visual field has been carefully examined, and the result checked six months after apparent recovery.

SPONTANEOUS CURE OF IDIOPATHIC RETINAL DETACHMENT. DR. G. MAZZATINI, Rome, Italy.

In three cases of myopic detachment with tear in which obturation of a retinal tear took place and the retina quickly reattached Dr. Mazzatini observed that spontaneous cure is quite possible. The obturation was due to an adhesive choroiditis. These cases corroborate Gonin's conception of the pathogenesis and his operative procedures, but they should not induce one to defer surgical intervention for more than three weeks in the hope of spontaneous reattachment, as retinal function is bound to deteriorate.

RETINAL DETACHMENT AS A RESULT OF INDIRECT INJURY. DR. M. DAVIDSOHN, New York.

Dr. Davidsohn examined forty-five patients from the point of view of the workmen's compensation law and was not able to confirm Gonin's findings of objective differences of appearance and location of the tears and the detachment in directly and indirectly caused detachments. In the indirect group, which includes cases without myopia, senility and myopia predominate. The time between trauma and detachment is generally less than two weeks. The group caused by direct injury shows an interval of more than three weeks in the majority of cases. Forty per cent of the operations according to Gonin's method were partially or completely successful. Eighty per cent of the patients not operated on became blind, and in 10 per cent the blindness was bilateral. In

place of the two categories, direct and indirect, and the antithesis of spontaneous and traumatic detachment, one must discriminate between domestic and industrial injury. Compensation should be awarded in all cases in which an injury has been shown and in which complaint is made of progressive clouding of vision in the two following days and detachment is found within two weeks.

SURGICAL MANAGEMENT OF RETINAL DETACHMENT. DR. J. LOPEZ ABADIA, Bilbao, Spain.

Dr. Lopez Abadia attaches little significance to closure of the retinal tear and considers the formation of adhesions between the retina, choroid and sclera of prime importance as the deciding factor.

APPLICATION OF ELECTRO-DIAPHAK TO THE TREATMENT OF RETINAL DETACHMENT. DR. J. LACARRÈRE, Madrid, Spain.

The distinguishing feature of this apparatus is the regulation of the current before operation. The active electrode is a universal handle to which blunt end-pieces can be attached for the purpose of parching the sclera (*Pergamentisierung*), according to Weve's method, or thread-like steel wires enclosed in a capillary tube insulated with glass. By pressure on a button of the handle, a spring is released, causing the ends of the wires to snap out of the tube. The advantages of this apparatus consist in the possibility of preoperative regulation of current and of depth of penetration by adjustment of the steel wires in the capillary tubes, extreme delicacy and accuracy of the punctures and coagulations, which for this reason can be made much more numerous on account of the exceeding fineness (0.18 mm.) of the steel wires, and good insulation. Diathermy coagulates rather than perforates, while thermo-puncture has just the opposite effects.

DETACHMENT OPERATION WITH ELECTRIC STRIP EXCISION OF THE SCLERA AND CHEMICAL CAUTERIZATION. DR. HILDESHEIMER, Berlin, Germany.

It is essential to bring about a marked irritation which will cause an extensive and progressive inflammatory process. This desideratum is not fulfilled by electrocoagulation, which has the further disadvantage that the inflammation does not really "get to work" until twenty-four hours later. The solid potassium point of Guist and of Lindner is very well suited to produce an artificial adhesion surface and may also, perhaps, act to bring about a certain amount of shrinking in the ocular tunics. Dr. Hildesheimer has further simplified Guist's method in that he actually excises a scleral strip 2 mm. wide by means of a platinum (wolfram) diathermy loop. In testing this procedure on living animals, it was possible to strike the sclera alone. In human beings, the operation meets with certain difficulties, which are, however, not insurmountable. In the experimental animals, hyperopia developed. So far, the operation has been performed for detachment. It is free from danger and has been followed by complete success in five cases.

DISCUSSION

DR. SOURDILLE: Arruga's conclusions as to the prime significance of a retinal, rather than a vitreous, pathologic process in the mechanism of detachment are important.

FURTHER DEVELOPMENT OF THE OPERATION FOR DETACHMENT WITH CAUSTIC POTASH. DR. GUIST, Vienna, Austria.

This article will appear in full in the *Klinische Monatsblätter für Augenheilkunde.*

DISCUSSION

DR. K. LINDNER, Vienna, Austria: Spontaneous detachment does not occur with a normal vitreous. With ocular movements the remnant of vitreous jelly swings to and fro. With certain rapid motions the retina may tear through where it is adherent to the vitreous, especially if it is in a state of cystic degeneration, besides, at these points. The loose flap is adherent to the vitreous and protrudes toward the interior of the eye. Accordingly, it may happen that a flap is actually torn out of the retina without causing a detachment. Shreds of degenerated vitreous can never produce, by their throw and impact, a flap tear of this kind. For this a certain mass, that of the remaining compact vitreous body, is required. I have never claimed that the tear is produced by shrinking of the vitreous, but the vitreous certainly shrinks after a tear occurs. If there is any fixation of the strand or formation of folds in the retina, the usual operative procedures offer no prospect of success, but improvement can be obtained by decreasing the volume of the globe. I have operated on seven eyes by means (with one exception) of annular scleral resection carried out in two separate sessions. In cases of hole at the macula, undermining is the only intervention to be considered. This has been performed in nine cases with seven cures. In other cases I use electrocoagulation. In regard to the frequency of recurrence, there appears to be no difference between Gonin's and Guist's operation (forty-nine cases with nine recurrences against thirty-two cases with six recurrences).

STATISTICS ON SIXTY-FIVE PATIENTS OPERATED ON BY GUIST'S METHOD. DR. R. CASTROVIEJO, New York.

The results obtained at the Medical Center of Columbia University with Gonin's method were comparatively discouraging, not more than 15 per cent of cures being obtained. Of sixty-five patients operated on according to Guist's method, 40 per cent were cured and 14 per cent slightly improved. In three patients who had previously been operated on unsuccessfully by the Gonin method, reattachment took place after the Guist operation. In the discussion, Dr. Castroviejo reports in detail thirty-nine cures in one hundred cases.

PRELIMINARY MEASURES IN THE OPERATIVE MANAGEMENT OF RETINAL DETACHMENT. DR. J. GONIN, Lausanne, Switzerland.

If for any reason one does not proceed at once to operation after noting the presence of retinal detachment, one must by all means insti-

tute treatment and take steps to prevent the operative prognosis from becoming less favorable. The first duty is to locate the tears and register them on a diagram, or better still to mark the meridian with india ink on the sclera at the opposite margin of the cornea. The eye must be immobilized completely and at once. This is much more important than mere rest in bed of the body as a whole. Lying flat is of advantage only in case the detachment is located in the upper half of the fundus. If the detachment has started below, the head is to be kept as nearly as possible in a vertical position and the patient may sit up on a sofa, or even walk about with due precaution, with less danger than he would incur in lying supine. With the head erect there is a considerable possibility of the detachment being displaced downward, freeing the region of the macula. Likewise, it is expedient to have the head inclined to the right, if the detachment is on the right, and, contrariwise, to the left side. Removal of the subretinal fluid by puncture leads at once to transient improvement, but only exceptionally to recovery. Unless the physician is prepared to proceed with the operative treatment the punctures should certainly not be carried out in case of recurrence.

DISCUSSION

DR. WEEKERS: In order to immobilize the globe, a step which is of the greatest practical importance, I use retrobulbar injections of a 1:500 solution of oxycyanate of mercury (1 cc. in the region of each external rectus muscle). The result is a severe inflammatory reaction which has a favorable influence on the detachment. Slight exophthalmos is produced, and the globe remains immovable for several weeks. This clears up gradually later, and finally disappears completely without any trace of damage to the eye. In the same way, I paralyze the levator palpebrae superioris muscle.

DR. ARRUGA, Barcelona, Spain: Discovery of the puncture point is facilitated by the steam bubbles which are generated in the interior of the eye in the course of the diathermy operation. Vitreous traction plays no part and is not a factor in the pathogenic mechanism. (Dr. Arruga discussed the constitutional condition and treatment.) The vitreous is not actively concerned. As it is a nonvascular structure it may take part, passively, in diseases of the ciliary body, but these are not usually of great importance.

DR. IMRE, Budapest, Hungary: Weve's method is excellent in case of small holes without actual "tears." If there is traction from vitreous strands, electrocauterization is of value to destroy the tissue connection between the vitreous and the retina. The diathermy-coagulation needle must not be allowed to penetrate too deeply. Even with microcoagulation, according to Weve's method, this may lead to secondary formation of holes or strands, as can the potassium hydroxide procedure, even after a lapse of months.

DR. GENET: I have used diathermy, independently of other authors, for one and one-half years.

ETIOLOGIC SIGNIFICANCE OF TRAUMATISM IN SPONTANEOUS DETACHMENT OF THE RETINA. DR. J. P. JEANDELIZE and DR. R. BAUDOT, Nancy, France.

Dr. Jeandelize and Dr. Baudot lay stress on the frequency of injury, even of slight degree, and on its importance, especially from a medico-legal standpoint, in cases of what may be only apparently spontaneous detachment.

OBSERVATIONS ON THE BEHAVIOR OF THE CICATRIX PRODUCED BY PERFORATING THERMOCAUTERIZATION AND ITS SEQUELAE, WITH SPECIAL CONSIDERATION OF THE INFLUENCE OF THE ORGANIC CONDITION. DR. D. SABBADINI, Rome, Italy.

Dr. Sabbadini has observed systematically the appearance of the scar after Gonin's operation, especially in regard to color, with the idea of establishing some relationship between this clinical feature and the posisible complications and sequelae. Immediate complications, i. e., those which take place within the first thirty days, have no great significance. The later secondary changes are much more important. In forty patients operated on according to Gonin's method, it was found that the scar is flat and extensive but smooth with a good deal of pigmentation in cases of syphilitic arteriosclerosis, and in those in which syphilis is suspected. There are no traction grooves or folds, and no preretinal hemorrhages later. Tuberculous patients present much more irregular scarring but with very little or no pigmentation, and frequently with glassy traction threads. In tuberculous children there is often a delayed fold formation which augurs ominously (recurrence of tears) and after from thirty to sixty days preretinal hemorrhages develop, with an unfavorable course. One may have to count on and deal with several tears in cases of extensive cystoid degeneration and in those with slight tears but with a marked tendency to neoplastic proliferation. Here, less energetic measures are advisable.

CLINICAL CASES OF OPERATIVE CURE IN DETACHMENT. CRITIQUE OF MODERN SURGICAL PROCEDURES. DR. P. BUFIL, Barcelona, Spain.

Recovery with full vision and complete restoration of the field is very unusual. When the tear could be located, Gonin's method was used; otherwise, that of Sourdille. Tuberculin was used in the postoperative treatment and as a follow-up measure.

TREATMENT OF DETACHMENT WITH TRANSSCLERAL DIATHERMOCOAGULATION WITH THE "PYROMETRIC ELECTRODE." DR. L. COPPEZ, Brussels, Belgium.

This instrument is a combination of the usual electrode with an electric thermometer, allowing measurement and registration of the temperature at the point of cauterization. In this way one can administer carefully measured and always identical amounts of coagulation. A temperature of 80 C., which is attained by a 2 mm. electrode after being in action for twenty seconds, is the best suited to produce a chorioretinal adhesion reaction without injury to the vitreous or sclera. Twenty cases are reported.

OPHTHALMOSCOPIC REGISTRATION, BY DRAWINGS, OF RETINAL DETACHMENT. DR. M. AMSLER, Lausanne, Switzerland.

Dr. Amsler demonstrates several examples and warmly recommends his methodical and simple procedure for drawing and registering the countless clinical pictures seen with the ophthalmoscope in detachment. A complete ophthalmoscopic drawing, a veritable topographic survey of the fundus both in surface extension and in depth of the eye, shows the state of papillary luster and the condition of the retina, the choroid and the vitreous as a whole and in detail to the extreme periphery of the ophthalmoscopic field. These accurate ocular data form the basis of diagnosis, of prognosis and of any therapy which aims to be something more than empirical. To give a strict account to oneself and, above all, to others as to just what has taken place in an eye that has been operated on is possible only by means of ophthalmoscopic drawings, and these must take the place of insufficient notations and verbal communications.

DETERMINATION OF RETINAL TEARS IN DETACHMENT. DR. P. TOULANT, Algiers, Algeria.

Dr. Toulant describes a simple method, requiring no special apparatus, to calculate the distance of the tear from the limbus. The patient, whose head is on a level with that of the oculist, and at the usual known distance, follows with his eyes a marker which an assistant carries along a vertical rule until the tear is seen in the center of the ophthalmoscopic field. From the rectilinear distance between the optical axis and a line through the tear in the examined eye, a simple calculation allows the angle of aberration between these two axes to be computed and determines accurately the value of the arc separating the tear from the macula in degrees of angle and finally in millimeters.

DIATHERMY IN THE TREATMENT OF DETACHMENT AND OF BUPHTHALMOS. DR. H. WEVE, Utrecht, The Netherlands.

1. Dr. Weve reports statistics on diathermy operations for detachment performed in 1932. The results were very satisfactory in cases of not more than two months' standing (more than 80 per cent of complete cures) but much less brilliant in older cases, especially in those complicated by aphacia (in which operation was performed, as a routine, by Gonin's method), among which there were only 33 per cent cures. The results in cases of previous dialysis (disinsertion) are particularly excellent, as all of the patients treated were cured. Dr. Weve calls attention to reactions of an anaphylactic nature observed in cases of repeated operations, and discusses certain technical details.

2. The observation that treatment by diathermy, even when successful, is followed by a prolonged period of markedly lowered intra-ocular tension induced Dr. Weve to try out this procedure in cases of buphthalmos in which other methods had failed. He gives details of the results and the technic.

TREATMENT OF IDIOPATHIC RETINAL DETACHMENT BY OBLITERATING FORTIFIED IGNIPUNCTURE. DR. F. CHAVARRIA, Calahorra, Spain.

Dr. Chavarria marks the position of the tear on the sclera and surrounds it with a series of cautery pricks down to the level of the choroid but without perforation, finally concluding the operation with perforating cauterization at the tear. At the point to be perforated the galvanocautery is applied a number of times in order to get the vitreous to detach.

SURGICAL TREATMENT OF RETINAL DETACHMENT. DR. J. C. BRAGE, Seville, Spain.

Dr. Brage operated on three patients with idiopathic detachment according to Sourdille's method and on six according to Gonin's method. In the first three cases there were one failure and one complete and one partial success. In the second (Gonin) group, there were three complete successes, one improvement and one failure.

RESULTS OF OPERATIVE TREATMENT IN DETACHMENT: REPORT ON SEVENTY-NINE CASES. DR. F. TERRIEN, DR. P. VEIL and DR. M. DOLLFUSS, Paris, France.

Patients with fresh detachments were operated on according to Gonin's method, at times with modifications; the others, by the methods of Guist, Lindner, Paulique and Sourdille. Thirty-one, i. e., 37 per cent, were cured. Of the fifty-seven patients with recent detachment with tear, forty-eight were operated on according to the Gonin method; twenty-two (45 per cent) were cured, nine were improved, in seventeen the operation was a failure and four had relapses in the first nine months after operation. Five patients were operated on according to Paulique's method with cauterization encircling the tear; two were cured, and three are still under treatment. Two were operated on by Guist's method; one was cured, and one improved. In two patients Sourdille's operation was used; in both a failure resulted. Of twenty-two patients without a tear, 20 per cent were cured. Of the three patients operated on by the Paulique method, one was cured and one improved moderately and one is still under treatment. Of the fifteen on whom the Guist operation was used, four were cured, one had a relapse after three months, three were improved and in nine the operation was moderately successful. For four patients the Sourdille operation was used; in two instances it was fairly successful, and one patient was cured and one improved. Finally, seven cures followed the subconjunctival injection of a 1:1,000 solution of mercuric cyanide, and one cure, the subconjunctival injection (by mistake) of a 1:100 solution of the same drug.

CATARACT AFTER IGNIPUNCTURE FOR DETACHMENT. DR. N. BLATT, Bucharest, Hungary.

This late postoperative complication consisting of lenticular opacities may be due to various causes, as noted by Dr. Blatt in four cases and confirmed by experimentation on rabbits. The nearer to the limbus the

site of ignipuncture and the deeper and more prolonged the cauterization the greater is the danger of subsequent cataract and the more rapid its onset. The lenticular opacity starts axially at the posterior pole of the lens. The danger of causing cataract should not, however, deter one from performing this operation for detachment.

OPERATIVE TREATMENT OF DETACHMENT. DR. C. SHAPLAND, London, England.

Dr. Shapland gives the results of three years' work at the Royal London Ophthalmic Hospital (Moorfields) including thirty-three operative cases, with details of diagnosis and surgical procedure and statistics of results, for which the reader is referred to the original paper.

RETINAL DETACHMENT. DR. B. PARANJPE, Bombay, India.

Operations according to Guist's method gave the following results: (1) complete success on an experimental animal (dog); (2) restitution of light perception and projection throughout the entire visual field in a detachment of eight years' standing; (3) complete success with good vision in another case.

TREATMENT OF RETINAL DETACHMENT BY MULTIPLE DIATHERMY-PRICKING AND ITS RESULTS. DR. ŠAFÁŘ, Vienna, Austria.

Dr. Šafář reports on his well known method, which he has applied so far, to forty eyes. Reattachment occurred in 60 per cent. The best results are attained in fresh cases of myopia and in those following injury by blunt violence.

INTRA-OCULAR FIXATION OF RETINAL DETACHMENT. DR. F. POYALES, Madrid, Spain.

Dr. Poyales has fashioned gold needles which correspond to the curvature of the sclera. With these he penetrates the sclera, goes through into the vitreous, draws the retina back into contact with the choroid and causes both membranes to adhere closely to one another by means of a clip which holds the needle in place. The needle is in connection with the diathermy current. The effects were tested out thoroughly on experimental animals and confirmed by histologic examination. The needle is introduced through a small incision made with a Graefe knife. This operative procedure allows one to convince oneself that the right region of the retina is being attacked before the current is turned on and the circuit closed.

DETACHMENT AND TUBERCULOSIS. DR. P. LLUESMA and DR. C. CHORNET, Valencia, Spain.

Dr. Lluesma reports a case of detachment in a tuberculous subject. Treatment with tuberculin resulted in improvement.

CONSIDERATIONS ON THE PATHOGENESIS AND THERAPY OF RETINAL DETACHMENT. DR. M. MARQUEZ, Madrid, Spain.

The retina is normally held in position by the eccentric (centrifugal) pressure of the vitreous and by the intimate union of the prolongations

of the pigment epithelium and the outer portions of the rods and cones. Previous pathologic changes in the tissue elements which contribute to this intimate "copulation" are the deciding factor in the development of detachment. In consequence of the disconnection of tissue elements a fluid is produced which is not the vitreous but different from it physically and chemically. Tears are not invariably present. The percentage of cures has been greatly increased by recent advances in operative procedures, but good results were attained formerly too. Dr. Marquez has himself several cases of complete cures, one of twenty-five years' standing. Salt solutions are very helpful, but only if they are markedly hypertonic (from 20 to 25 per cent) and are injected far back under Tenon's capsule. The usual subconjunctival injections of weak (from 2 to 5 per cent) salt solutions into the anterior segment are painful and useless. The immediate results with hypertonic solutions are generally strikingly favorable. Perforating operations increase the probability of cure, especially when combined with deep "intratenonal" injections of a hypertonic solution. The puncture is effected at the deepest point of the detachment pouch (*Beutelhoehe*).

Is Rest Treatment of Appreciable Value in Detachment? Dr. E. Martinez, Madrid, Spain.

Dr. Martinez's experiments have demonstrated that there is very little initial difference in density between the retina, the vitreous and the vitreous filtrate but that this difference increases noticeably if the retina is brought in contact with hypertonic solutions of sodium chloride, which increase the density of the retina from 1.03 to 1.09. It has also been shown that a vitreous which has rested for twelve hours on a wrinkled retina is not able to smooth out the folds in this membrane. These facts would seem to prove sufficiently that rest of the eye alone, no matter how complete, is not enough to bring about a cure of detachment. Immobilization of the globe may be valuable in connection with operative treatment, especially with those procedures which bring the retina into contact with hypertonic solutions of sodium chloride and so increase the density of this tissue.

Inadequacy of Medical and Surgical Procedures in the Treatment of Retinal Detachment. Dr. T. Barraquer, Madrid, Spain.

The operations, one and all, do not do away with the causal factors, bring about only temporary "episodic" improvement and do not cure the disease or prevent recurrence. Appropriate action in the individual case depends on accurate indications. The intra-ocular tension gives the most definite and important information:

1. Soft eyes. There is to be no abstraction of fluid, either active or passive, from the interior of the eye. This would merely allow the retina to reattach at one point without in any way preventing its separation at some other point. On the contrary, one must contrive to harden the eye in some way, as by cauterization of Schlemm's canal or by the recently advised subconjunctival injections of hypotonic solutions to bring about hydration of the vitreous with consequent edema which

allows it to fill the interior of the globe more fully and thus raise the intra-ocular tension. At the same time, one must use medication to lower the blood pressure and thus favor resorption of the fluid under the detachment. This subretinal fluid in detachment is never vitreous. How could the vitreous make its way eccentrically and centrifugally through a tear which it had previously caused by the directly opposite mechanism of centripetal retraction to the point of tearing the retina?

2. Hard eyes. The subretinal fluid may be evacuated, as by simple puncture, or by Gonin's or Sourdille's procedure, but always with careful observation and control of intra-ocular tension, so that the latter may be brought up or down to the normal as the case may be, whenever necessary.

THERMOPUNCTURE AND CHEMICAL OR DIATHERMIC CHOROIDITIS ADHAERENS. DR. SOBHY BEY, Cairo, Egypt.

Dr. Sobhy Bey discusses the advantages and disadvantages of Gonin's procedure and recommends the Arruga and the Foster Moore type of needle. He has had no success with Sourdille's method. He was able to close macular tears by the Guist or Lindner procedure.

DISCUSSION

DR. S. MARTIN: I have tried various procedures but have had permanent successful results only when the tear has been closed up. I think that the Weve operation is the best.

DR. GALLENGA, Parma, Italy: I have shown experimentally that bits of tissue hardened in alcohol or a dilute solution of formaldehyde and allowed to heal in subretinally and intrachoroidally lead to the formation of adhesions between the retina and the choroid without any marked inflammatory reaction.

DR. RUBBRECHT, Bruges, Belgium: Sutures passed through the retina in the region of the detachment cause firm adhesions. The sutures remain in situ for two days. I have operated on two patients successfully by this procedure.

DR. STRAMPELL: I recommend the subretinal injection of blood plasma.

DR. SPECIALE PICCICHÉ, Rome, Italy: I have observed delayed reattachment of the retina in the course of days in association with a slow, progressive closure of the tear.

DIAGNOSIS OF RETINAL DETACHMENT. DR. L. PAVIO, Buenos Aires, Brazil.

Dr. Pavio presented sixteen ordinary photographs of the fundus and twelve stereoscopic views showing pathologic changes which affect unfavorably the outcome of operative procedures and offer a bad prognosis, such as folds under the vessels, "sharp," firm retinal folds, induration of the margins of the tear and vitreous adhesions.

(To be concluded)

FRENCH OPHTHALMOLOGICAL SOCIETY

Forty-Sixth Meeting, Paris, June 26-29, 1933

TRANSLATION BY DR. PERCY FRIDENBERG, NEW YORK

A NEW SLIT LAMP WITH HOMOGENEOUS ILLUMINATION. DR. LEMOINE and DR. VALOIS, Nevers.

It is well known that slit lamps of the usual model do not give, when in the Vogt position, perfect illumination of the region under examination unless focusing is rigorously exact. If the registering of the lamp is not perfect, one sees a series of bands, alternately light and dark, which interfere with the examination. If, on the other hand, one wishes to illuminate a broad surface, one has to give the lamp the Gullstrand position and in that case it is focal illumination which causes the disturbing bands to appear. In order to do away with these inconveniences, the authors have had made a bulb in which the light source is not a filament rolled into a spiral, but a tungsten filament. The light obtained with it is perfectly homogeneous. In the new lamp of Lemoine and Valois the condenser, now useless, has been done away with, so that there is no longer either Vogt or Gullstrand position. The practice of biomicroscopy is accordingly simplified and illumination improved.

CONGENITAL PARALYSIS OR ABSENCE OF ACCOMMODATION. DR. SEDAN and DR. ROUX, Marseilles.

Three young men, brothers, were observed who had no accommodation whatever and could read only with convex lenses, sph. + 4 D. They were not hypermetropic and distant vision was reduced by the addition of even convex sph. + 0.75 D. There was no previous history, either personal or familial, of syphilitic disease, diphtheria or diabetes. An only sister was normal. Neurologic examination yielded negative results. Aside from the accommodation, all ocular functions were normal, except that the pupillary reflex to convergence accommodation was completely missing. Atropine and physostigmine had no influence on accommodation. The underlying factor was probably a congenital lack of development of the ciliary muscle rather than a congenital partial paralysis of the third pair of nerves, as the motility of the iris was unimpaired. These congenital paralyses have been observed in cases of aniridia, but isolated ciliary aplasia is exceptional.

TREATMENT OF MARKED ECZEMAS OF THE LIDS. DR. TERSON, Paris.

In these eczemas, which are so distressing in adults, in the acute stage silver nitrate in from 2 to 3 per cent solution is used, at first, two or three days in succession. A vegetarian diet is prescribed with free use of fluids, laxatives, diuretics and hypnotics. A complete clinical examination is made, with analysis of the blood, urine, etc., and the appropriate therapeutic management is instituted. Powders of sulphur, charcoal and magnesia are taken after meals. No local washes are used. The lids are lightly mopped with lukewarm water containing a small amount of coal tar solution or sulphonated bitumen. After the areas have been made dry, the nitrate of silver is given at greater intervals.

Pastes are then used of water and zinc oxide or talcum powder, together with 2 per cent of ephedrine, which lowers congestion and produces a remarkable sedation. Finally, fatty preparations may be tried, such as a salve containing 1 per cent silver nitrate. Yellow salve (ointment of yellow mercuric oxide) should not be used in adults. The diet is the same as for a constipated "bilious" subject with renal trouble. Alkaline or acid phosphate salts are given for acidosis or alkalinosis, respectively. In children, after the silver nitrate, one may try ointment of yellow mercuric oxide, 2 per cent, often with rapid cure. Sea air and sea bathing are to be avoided. The waters may be taken at a sulphur and arsenic spa such as Saint-Honoré.

INFREQUENT COLD WET APPLICATIONS IN OPHTHALMIA NEONATORUM. DR. CHARLES ROCHE, Marseilles.

Within the last fourteen years Dr. Roche has treated sixty-three cases of gonorrheal conjunctivitis of the newly born without seeing ulceration of the cornea develop in a single case. The serious nature of this complication is well known and generally recognized. Roche attributes its appearance to previous traumatism of the anterior layers of the cornea in the course of too frequently repeated irrigations attended by more or less skilful instrumentation. He uses only one wet application a day. The lids are separated with the fingers or, if necessary, a lid elevator may be used, with care not to touch the cornea. The culdesacs and the folds of the fornix are cleansed with a few cubic centimeters of a solution of potassium permanganate, 1:5,000, which is squeezed out of a sterile absorbent cotton "wipe" and allowed to fall on the eye from a height of 1 or 2 cm. The culdesacs are now dried with a tampon, well squeezed out. With the face of the child held horizontal, 2 or 3 drops of a solution of silver nitrate, 2.5 per cent, freshly prepared and kept from the light, are instilled "frankly." After this, the eye is lightly irrigated with the potassium permanganate solution and the conjunctival sacs are partially filled with a mildly antiseptic salve.

CORNEAL LESIONS AND THE POSTERIOR CERVICAL SYMPATHETIC SYNDROME. DR. H. VIALLEFONT, Montpellier.

Viallefont reports three observations of obstinate relapsing corneal ulceration with lowered corneal sensibility associated, by way of a cervical arthritis, with a posterior cervical sympathetic syndrome. In the opinion of the author, this is due to a circulatory disturbance in the region of the trigeminal centers, and the condition is one of a neurotrophic "trigeminal" keratitis. In two cases, treatment of the accompanying arthritis brought about lasting cure. The author advises a search for the aforementioned syndrome in all cases of "hypo-esthetic" keratitis of indeterminate origin.

EXPERIMENTAL AND HISTOLOGIC STUDIES OF CORNEAL INCISIONS BY THE ELECTRIC KNIFE. DR. H. VILLARD and DR. C. DEJEAN, Montpellier.

The electric bistoury easily incises the cornea, producing a wider and more irregular section than does the Graefe knife. Histologically, the

lesions produced are distributed in four concentric zones: 1° zone of bursting or explosion, in which the tissues are destroyed; 2° zone of coagulation, in which they are amorphous and agglomerated to a brown crust which carpets the wound; 3° zone of elongation, in which the histologic elements are recognizable but deformed and stretched out; 4° zone of normal tissues. If the section is sufficiently deep to reach the neighborhood of the anterior chamber, the underlying iris is swollen and coagulated and its vessels are obliterated over a certain area. The endothelium of Descemet's membrane also swells up and desquamates. All of which goes to show that the fluid media of the eye, excellent conductors of the waves of a high frequency current, are able to carry to a distance the coagulation effects of diathermy. Sections of rabbits' eyes, two, four, eight, fifteen, thirty and fifty days after cauterization, give evidence of a rapid cicatrization of epithelial and connective tissue. But all surgical intervention should, in the case of the eye, be controlled with skill and care.

Reduplication and Development of Descemet's Membrane. Dr. Redslob, Strasbourg.

In the staphylomatous cornea of an eyeball affected by an old parenchymatous keratitis Redslob observed a neoformation of Descemet's membrane. The proliferation doubled the original tissue and lined the primitive membrane. At certain points, this lining was separated from the primitive membrane by a cavity; at others, the double membrane was attached only at one end to the primitive membrane, the other end penetrating obliquely into the anterior chamber and ending in a sort of looped thickening. All these newly formed membranes were edged with an endothelium of normal appearance. These data are most unusual; the observation, very rare. From the findings of embryologic research, as well as from the data of experimentation and those of pathologic histology, it follows logically that it is the endothelium which actually produces or, as the author says, elaborates Descemet's membrane. These cells of the posterior epithelium have a very complex structure and fulfil multiple functions. The membrane might well be a product of secretion, but it might just as well, as appears on histologic examination of the cornea in question, be the result of cellular action, by intermediary agency of ferments (Nageotte), on the fibrinous substance of the neighboring structures. Before being definitely formed, the membrane seems to pass through a state analogous to that of connective tissue fibers. By a process of hyalinization these fibers are then transformed into homogeneous, regularly refracting and hence transparent substance.

Primary Preeruptive Herpetic (Zostérienne) Adenitis in a Case of Ophthalmic Herpes. Dr. Cuenod and Dr. Nataf, Tunis.

The authors report a case of this disorder in which the cutaneous phenomena (erythema and eruption) were definitely preceded by adenopathy. This is an original observation which confirms the existence of a primary, preeruptive adenitis in herpes febrilis as claimed by Barthelemy in 1892. Observations of that adenopathy in herpes zoster

support the hypothesis of the direct and simultaneous influence of a posterior poliomyelitis, the original lesion in herpes zoster, on the lymph glands and on nutritional processes in the skin.

TREATMENT OF FOLLICULAR (OCULAR) TUBERCULOSIS WITH THE METHYLIC ANTIGEN OF NÊGRE AND BOQUET. DR. BEAUVIEUX, Bordeaux.

The author refers to the concept of Ranke in the determinism of ocular tuberculosis, according to which the latter is to be considered as a superinfection in an allergic tuberculous subject, and shows the value of antigen therapy in the various clinical forms of this disease. Among the numerous vaccines which have been recommended from time to time, the methylic antigen of Nègre and Boquet, it appears, deserves to be adopted by a large number of practitioners on account of its easy administration and the absence of any danger in its use. It is a valuable diagnostic and also therapeutic agent which is often efficacious. The reports which have appeared up to the present time relate mainly to the use of this methylic antigen in the afollicular forms of tuberculosis of the eyeball and show very encouraging results. The author, following Onfray, Rousseau and Spillmann, gives the clinical details of two of his own cases of severe follicular tuberculosis of the anterior segment, cured in two years by means of antigen therapy, after enucleation had been advised in one case. On the other hand, in cases of conglomerate, massive forms of tuberculosis, with threatening perforation of the globe, enucleation maintains all its preference and rights, less as a therapeutic procedure than as a measure of desperation. The author believes in the real value of the methylic antigen of Nègre and Boquet in those forms of ocular tuberculosis of an attenuated type, according to Leber, a value which animal experimentation alone, in all probability, will enable one to gage accurately.

REPORT: FOLLICULAR CONJUNCTIVITIDES. DR. MORAX.

The generic term of follicular conjunctivitis is applied to a series of involvements of the ocular mucosa which express themselves histologically by the formation of nodules in the thickness of the conjunctival mucosa but which, as a matter of fact, represent different diseases. Clinical study with animal experimentation has put one in a position to fix the limits of these diseases and to bring out certain points in their etiology. The reporter has taken as a basis of his study the form of follicular inflammation known as swimming-pool conjunctivitis and observed quite commonly now that "piscines" or basins for large numbers of swimmers in a comparatively small area have become so numerous in many countries. But this disorder, as the reporter points out, existed before swimming pools were in vogue or even known, and, furthermore, it is not necessary to visit one in order to get this disease. It is very often unilateral and is characterized by marked diffuse swelling of the lids, with redness of the conjunctiva of the fornix folds and later of the tarsal conjunctiva. There is more or less discomfort without abundant secretion. After several days the surface of the mucosa shows small lumpy prominences, the follicles. The condition may last

for from two to three months, but clears up without complications or sequelae. Experimental inoculation of human conjunctivae has shown that this disease is contagious. The reactive symptoms appear after a latent period of from six to seven days. The disease can also be transmitted to monkeys (Macacus rhesus) and chimpanzees. The rôle of the swimming pool in the diffusion of the infection has been definitely proved, but up to the present time it has not been possible to demonstrate clearly just how the infection is conveyed from an infected subject to a sound one. One has to deal with a special infection the underlying causal agent of which has as yet not been demonstrated. The report considers in detail the question of epithelial inclusions, or Prowazek bodies. The data relative to this factor merely serve to complicate still further the differential diagnosis between follicular conjunctivitis and trachoma. This diagnosis can rest securely only on the basis of the different clinical evolution and course of the one condition and the development of complications and sequelae in trachoma. In the course of his remarks on diagnosis the reporter takes into consideration other forms of conjunctivitis with follicle formation and discusses briefly: conjunctival folliculosis, a benign disturbance which does not give rise to any ocular reaction of moment; acute follicular conjunctivitis, Beal type, which generally runs its course in from two to three weeks; chronic follicular conjunctivitis, which experimental study has proved to be contagious and which may last for more than a year; toxic, or rather chemical, follicular conjunctivitis, caused by intolerance of atropine or physostigmine in specially sensitive or predisposed subjects. Morax then points out certain diagnostic difficulties in connection with spring catarrh, tuberculous conjunctivitis and a special form of circumscribed conjunctival infiltration with benign course and adenitis. The treatment of swimming-pool conjunctivitis is purely symptomatic.

PECULIAR CASES OF FOLLICULAR CONJUNCTIVITIS. DR. C. DEJEAN and DR. T. TEMPLE, Montpellier.

The authors report on an epidemic of follicular conjunctivitis in a boarding school for young girls. They also present their observations in three cases of chronic hypertrophy of the conjunctival follicles taking on clinically as well as under the slit lamp the form of small semitransparent ovules or opaline, sausage-like rolls. Bacteriologic examination gave no useful data. Biopsy of a bit of mucous membrane showed in every case a lymphoid hypertrophy with infiltration by lymphocytes and plasmocytes and finally, in two cases, a quite extraordinary development of the muciparous glands or, rather, a mucoid degeneration of the epithelial cells.

PUPILLARY REFLEXOMETRY. PRESENTATION OF AN ORIGINAL APPARATUS (PROJECTIONS). DR. BUJADOUX, Lyons.

The author, with Kofman, presented a new apparatus permitting graphic registration of the photomotor reactions of the pupil. A series of light stimuli of gradually increasing intensity are used which allow the diameter of the pupil to be noted in progressively decreasing figures. When these are reduced to a graph there results a curve in the form

of an imperfect exponent. This curve may be modified by physiologic factors or by disease. Thus one may preserve the record of results of experiments or of clinical observations, and easy comparisons can be made in one and the same subject on the pupillary response to light. The entire range of pupillary contraction can thus be studied dynamically. The authors have finally established the existence of a flat, "table-land" graph in the first phases of pupillary contraction, and this they have found to agree with the cytologic modifications of the cerebrospinal fluid in numerous cases of syphilis of the central nervous system. Here one is probably dealing with a pre-Argyll Robertson phase of pupillary disturbance.

CONTRIBUTION TO THE STUDY OF DIABETIC CATARACT BY MEANS OF THE SLIT LAMP. DR. J. BADOT, Jumet.

Three factors seem to dominate in diabetic opacification of the crystalline lens: (1) the appearance of subcapsular opaque masses, at first isolated, and becoming confluent later on; (2) the appearance of clear clefts by separation of the sutural lines of the lens fiber mass; (3) the presence of vacuoles which have their seat predominantly in the superficial layers of the lens capsule.

TOTAL EXTRACTION OF CATARACT IN TWO STEPS (LANTERN SLIDES). DR. C. DEJEAN, Montpellier.

This procedure consists, according to the author, in a first step to extract the lens nucleus and a second to "extirpate," i. e., withdraw completely, the lens capsule from the interior of the eye. A fine, sharp, hooklike cystitome allows him to make a discission with a triangular flap. After letting out the nucleus, a smooth grasping forceps, without teeth but with broadened spatulate tips, allows this flap to be seized and firmly held, making tangential traction, and carefully limited and control pulls on the fibers of the zonule, which gives way a little at a time in small "paquets." The procedure appears to be simple enough, but the author warns that it calls for the "sanction" of experience.

TWO SAFETY PROCEDURES IN TOTAL EXTRACTION OF CATARACT. DR. DE SAINT-MARTIN, Toulouse.

The first consists in a form of corneal suture recently devised by Dr. Gomez-Marquez of Barcelona. It proves extremely effective in preventing the escape of vitreous while the lens is being extracted with the forceps or with Barraquer's suction apparatus. It differs from the "classical" corneal suture of Kalt and the successive modifications of his procedure, in that, with one hand, alone, the assistant is able at any moment to turn down the corneal flap and bring the lips of the operative incision into contact. Several drawings illustrate more definitely the placing of the suture, the arrangement of the threads and the way in which the knot is tied. The author has applied it in more than 150 operative cases, without loss of vitreous. The second precaution consists in effecting a *prolonged* paralysis of the lids and of the orbicularis by juxtaperiosteal injections, according to the akinesia technic of Van Lint-Villard, of 6 cc. of a 2 per cent procaine hydrochloride solution

in 45 per cent alcohol. In this way one avoids any brusque squeezing of the lids during the early period of the healing of the wound while the cicatrix is still delicate, say for ten or fifteen days after operation. This procedure causes no inconvenience and is open to no objection.

TECHNICAL DETAILS OF INTRACAPSULAR EXTRACTION OF CATARACT (LANTERN SLIDES). DR. ARRUGA, Barcelona.

Certain details are of prime importance for the successful removal of the lens in the capsule. The tips of the capsule forceps should be advanced to the center of the pupil and then pushed, under the iris, to the lower part, to be applied and to rest against the anterior surface of the lens near its lower border. The forceps is now given an almost vertical direction and the tips of the branches are allowed to separate to 4 mm.; they are then pressed against the lens and closed so as to nip the capsule. Traction is now made on the forceps toward the center of the pupil, and zig-zag lateral motions made until with the aid of a semicircular squint hook the lens in toto comes out of the eye. If the cataractous lens is hard, firm pressure may have to be made with the hook. The percentage of cases of a lens successfully removed in the capsule is very high from the beginning, and if these instructions are followed one will be agreeably surprised in that one will never pinch the iris or dislocate the lens. The author presented a model of the forceps which he has perfected.

RETINAL DETACHMENT USHERED IN BY HYALITIS. DR. GENET, Lyons.

Genet presents three observations of detachment the first manifestation of which consisted in clouding of the vitreous which came on suddenly but was preceded in some cases by the appearance, a long time ahead, of clouds lasting only a short time and followed by clearing vision. The question arises whether in these cases of detachment beginning with hyalitis one has to deal with the results of a more or less profuse vitreous hemorrhage or with a flare-up of a choroiditis accompanied by marked clouding of the media. After the lapse of from two to four weeks the vitreous clears up. Its transparency reappears and may at times become almost complete. One now sees the classic retinal detachment with a tear at one point. The author believes that this form can be interpreted as follows: As shown by histologic examination cystic spaces may develop in the retinal tissue itself. Some of these cysts may become very voluminous and undergo an evolutionary spurt sufficiently marked to cause sudden rupture of the cyst into the vitreous. The latter is inundated with a turbid fluid which renders ophthalmoscopic examination impossible. After the lapse of a certain time, clearing of this medium allows one to detect a detachment of the retina with the usual characteristics and at one point of this detachment a retinal tear which corresponds to the point where the rupture of the cyst took place.

INSTRUMENT FOR IMMEDIATE LOCALIZATION OF RETINAL TEARS. DR. ROCHAT, Groningen.

The author presents and describes an instrument which makes it possible to fix the apparent site of a retinal tear at the very moment

at which it is seen with the ophthalmoscope. The latter is attached by three threads to a similar number of indicators which run along a scale. Their readings indicate at any moment the position of the ophthalmoscope and accordingly determine the position in space of the retinal image formed on it.

[NOTE.—In this way, not only can one find the tear again easily, but one can also point it out to another observer. To determine the apparent position of the tear in terms and degrees of longitude and latitude it is sufficient to replace the eye of the patient, automatically, by an arrangement of two dials placed perpendicularly to one another in the manner of a theodolite. On these dials one can read off directly the meridian in which the tear is located and the angle included between the axis of the eye and the incident ray which goes to the apparent place of the tear. This allows one to obtain in a few seconds the data necessary to localize the tear prior to operation.]

OBLITERATION OF RETINAL TEAR BY DIATHERMIC COAGULATION (LANTERN SLIDES). DR. GENET, Lyons.

Genet offered a projection of drawings of a retinal detachment before and after diathermic coagulation. The tear was completely obliterated by exudate. After two months the exudate was completely absorbed. Diathermic coagulation was repeated and was followed by suprachoroidal galvanocauterization. In spite of all this, a detachment appeared at another point of the retina. The author calls attention to his claim that he performed retinal diathermic coagulation as far back as and ever since 1931. It appears that one must be prepared in a certain number of cases to repeat the diathermic coagulation. There will also be occasion, not infrequently, to combine the various procedures. Diathermic coagulation does not seem to expose the patient, as does incision of the sclera with the bistoury or thermocautery, to massive hemorrhages into the vitreous. Diathermic coagulation is a simple procedure which should enable one to empty the subretinal fluid and then more easily find the site of the tear, and this intervention may be combined either with external applications of thermocoagulation or with suprachoroidal ignicauterizations over the tear or in its immediate neighborhood.

USE OF PYROMETRIC ELECTRODE IN THE TREATMENT OF RETINAL DETACHMENT. DR. L. COPPEZ, Brussels.

To attempt to measure the diathermic eschar in terms, or as a function, of the intensity of the current is an absurdity. It has been shown classically from of yore (d'Arsonval and others) that the thermic effects of high frequency currents cannot be measured in relation to their intensity. Accordingly, they must be measured directly. In scleral diathermic coagulation, in measuring the temperature of the eschar by means of the pyrometric electrode one is assured of a means of definite and accurate control which allows one to obtain therapeutic tissue reactions similar in every respect to a type lesion chosen, studied and experimentally determined in advance. The author then describes in detail his operative technic. The heat-registering and regulating elec-

trode makes the operation very simple and accurate and sure. The results show remarkable regularity.

OPERATIVE PROCEDURES IN RETINAL DETACHMENT. INDICATIONS AND RESULTS. DR. DE SAINT-MARTIN, Toulouse.

The author gives the clinical and operative details of thirty-eight cases of spontaneous detachment of the retina and the results obtained by him with the three forms of surgical technic recommended at the present time: thermopuncture of Gonin; chemical cauterization of Lindner-Guist; diathermic coagulation. All three owe their origin to the postulate of Gonin, to wit, the necessity of searching for, locating and obliterating retinal tears, the cause, or at least an important factor in maintaining and aggravating detachment. All three procedures received practically unanimous approval at the recent International Ophthalmological Congress at Madrid. The tear was found in almost all of these patients and those alone were cured in whom it had been completely obliterated. Nineteen operations after Gonin's method, that of obliterating thermopuncture, gave six cures. Six operations after Lindner-Guist, choroidal cauterization with potassium hydroxide, gave two cures. Finally, thirteen diathermic coagulations with the technic of Weve (Utrecht) gave eight cures. These results which are in accord with the principal statistics published up to date attribute the maximum of efficiency to the high frequency current, the most simple in application, least disturbing to the eye and best adapted to the majority of cases. They show to what an extent the prognosis has been improved in a condition hitherto considered as practically incurable.

SUTURE OF THE RETINA IN THE TREATMENT OF DETACHMENT (LANTERN SLIDES). DR. RUBBRECHT, Bruges.

Suture allows one to establish extensive and solid adhesions between the retina and the choroid without bringing about serious pathologic tissue changes in those membranes. In the region of the point of entrance and exit of the needles a proliferation takes place of cells derived from the episclera. In cicatrizing, this newly formed tissue "solders" the retina and the choroid together at the margin of the tear. Between these points the irritative reaction caused by the thread provokes a stretch of adhesive chorioretinitis. Rubbrecht presents a report of the two first observations of detachment treated by suture. In the first case, an extensive disinsertion was healed by two sutures running meridionally; in the second, he had to deal with a "vast" pouchlike detachment in the upper half of the globe, in an eye affected with nystagmus and aphacia. In both cases there was complete cure with perfect recovery and the patients got back their entire visual earning capacity.

CONDITIONS TO BE REALIZED BY OPERATIVE INTERVENTIONS IN DETACHMENT OF THE RETINA. DR. H. COPPEZ, Brussels.

All the surgical procedures at present in use have as their immediate object the barring out of the tear and the production of an adhesive chorioretinitis. A logically conceived operation must satisfy the follow-

ing requirements: 1. The operative procedure must be easily executed. 2. The operation must be capable of application to the greatest possible number of cases of detachment. 3. The choroiditis provoked must always be of the same intensity or, as Coppez says, "equal to itself." 4. The operative intervention should be directed mainly, if not exclusively, to the choroid. 5. The operative intervention should insure the closure or, at the very least, the "barring off" of the tear. 6. It is desirable but not indispensable for the retina and choroid to be brought into contact before the operation. 7. It is indispensable for the subretinal fluid to be evacuated in the course or at the finish of the operation and that continued off-flow should be assured during the following days. 8. The operative procedure must be not only curative but preventive. 9. The operative procedure must be capable of completion or renewal within a very short space of time. It would seem that the operation which at the present time answers these conditions best consists in diathermy with pyrometer control.

CONTRIBUTION TO THE PATHOLOGIC PHYSIOLOGY OF THE CENTRAL RETINAL VEIN (LANTERN SLIDES). DR. FRITZ, Mons.

It is possible to recognize *in vivo* to what degree a thrombosis of the central retinal vein is permeable. On compression (by pressure on the globe) the vein empties of blood more or less quickly and under greater or less degrees of compression. Continued pressure creates ahead of the thrombus a much more marked venous hypertension than in the region of a normal vein, complete vascular obstruction appearing to be favored by the thrombus. These data may enable one to prognosticate the possibilities of restoration of normal circulatory conditions and/or the danger of secondary glaucoma which would be the ultimate result, generally, of total obstruction of the central vein. They also aid one in interpreting the state of permeability of the vein, which is often abnormally reduced in the course of a chronic glaucoma. Finally, they establish the causal factors and to some extent the pathologic mechanism in the various forms of collapse (*écrasement*) or occlusion of the central vein with or without pulsation.

CERTAIN CELLULAR FORMS ENCOUNTERED IN THE COURSE OF DIVERS INFLAMMATORY CONDITIONS OF THE RETINA. DR. MAGITOT, Paris.

The elements in question are vacuolar cells resembling those which the author, in collaboration with Mawas, described in 1913 as observed in the vitreous of the human fetus in the third month. These cellular elements measure from 30 to 60 microns and have a small nucleus which stains well, surrounded by a crescent of protoplasm. As the vacuole increases in size it pushes the nucleus toward and against the wall of the cell and the cell takes on the appearance of a rounded bleb. These elements have been found in detachment of the retina, papilledema, glaucoma and albuminuric retinitis. They are located either in the vitreous in contact with the retina, or in the retinal fluid. The author has provoked the appearance of similar cells in the eyes of a rabbit which he subjected to thermopuncture. The cells appear to originate in the layer of bipolar cells and the ganglion cell layer. In all probability, they are neuroglia cells.

RADIOTHERAPY OF THE SUPRARENAL REGION IN THE TREATMENT OF DISEASE OF THE RETINAL CAPILLARIES. DR. ZIMMERN, DR. MAGITOT, DR. TILLE and DR. BRUNET, Paris.

The good results obtained by means of suprarenal radiotherapy in cases of obliterating endarteritis of the extremities led the authors to apply this form of treatment to senile retinitis and to the guttate softening (*ramollissement en gouttes*) seen with sclerosis of the retinal vessels. Of nine cases observed, two showed marked improvement, three a slight change for the better and four no result. In circinate retinitis this method of treatment appeared to be without effect, while in the milder cases in which the macula was involved it was efficacious and of evident benefit, as vision was decidedly improved. The irradiation should reach not only the suprarenal gland itself, but the paravertebral and prevertebral dense sympathetic plexus (*Feutrage*) as well, to the exclusion of the lumbar cord. To this end it is necessary to use a localization which is definitely decentered and lateralized in two circular and visibly symmetrical fields the inner tangent of which remains about 8 cm. distant from the median line. The fields are 10 cm. in diameter and straddle the twelfth rib. The tension is 130,000 volts. An aluminum filter 5 mm. thick is used. From 400 to 500 roentgen skin units is applied at each session and for each field. A series of four treatments is given, or from 1,600 to 2,000 roentgen skin units, spread over eight or ten days, and an interval of from fifteen to twenty days allowed to lapse before taking up the following series.

OVARIAN THERAPY IN RETINITIS PIGMENTOSA. DR. J. FRANCOIS, Mons.

The author has tried this form of opotherapy in four cases, using subcutaneous injections of 100 mouse units of a water-soluble female sex hormone daily. In the first case the visual field was appreciably enlarged, although neither vision nor light sense was improved. In the second case he was able to obtain a manifest improvement of vision and of light sense, but the field was only slightly influenced, if at all. In the third case, which was of very long standing and so not at all promising in regard to possible effects of treatment, he got no results. In the fourth case there was considerable gain in the extent of the visual field with definite improvement in light sense, but visual acuity was not increased. If after two months of this treatment one has seen no improvement, there is no further reason to hope for it. The treatment is interrupted when it gives no further improvement. When it is taken up again after one or two or at most four months, one runs the risk of losing whatever beneficial results had been gained before. Female sex hormone acts, when it does act, on the visual field, principally, and on the light sense. Visual acuity is affected to a much slighter degree. The restoration of the outlines of the field takes place in the order in which they were lost.

INFECTIOUS BILATERAL OPTIC NEURITIS IN THE COURSE OF EXANTHEMATIC FEVER OF THE MEDITERRANEAN LITTORAL. DR. AUBARET and DR. GUILLOT, Marseilles.

The authors report the first case of the nature indicated in the title, observed in a young man of 26, affected with *fièvre boutonneuse*, or

exanthematic fever of the Mediterranian coast. This disease, only recently discovered and studied, thanks mainly to the works of the Marseilles school, may, it appears, present other ocular complications than the usual inoculation conjunctivitis, described at the same time as the conjunctival inflammations ascribed to ticks, which were also noted at Marseilles.

Clinical Considerations on Atrophic Excavation of the Optic Nerve Without Intra-Ocular Hypertension. Dr. Baillart, Paris.

The author reports the observation of a case in which increased intra-ocular tension did not come on until two years after the beginning of the atrophic excavation of the nerve head. Both glaucoma and excavation have a vascular disturbance as their common underlying cause. If this disturbance involves the anterior segment the result is increased intra-ocular tension. If it is localized in the posterior segment the excavation alone appears. Insufficiency of circulation in both regions brings about increased tension with atrophic excavation, but these two clinical signs of a vascular disturbance do not necessarily appear at the same time.

Strabismus and Avitaminosis. Dr. Dor, Lyons.

The author reports on the observation of an infant whose mother and maternal aunt had both been affected with strabismus, and who itself presented a convergent squint at the age of 9 months. The child was treated with a vitamin preparation given in six daily milk bottle (*biberon*) feedings, and in the fourteenth month the strabismus completely disappeared. In cases of strabismus definitely installed for more than one year, treatment with vitamins gave no result but this is in keeping with Mouriquand's demonstration that vitamins are completely inactive in avitaminoses of long standing. In questioning the parents as to their method of nourishing squinting infants, Dor got the impression that most of these infants had been kept on an exclusive milk diet too long and had not eaten vegetables or yolk of egg until too late. It is not enough to give them lemon juice which contains the C vitamin. They also require vitamins A and D. Vitamin B is always present in sufficient quantity in the milk, but vitamin A, although present in sufficient amount for the development of a normal child, may be inadequate for the development of the visual apparatus in an infant with hereditary strabismus.

Interferometry. Clinical Results in Ophthalmology. Dr. G. Weill, Strasbourg.

The author gives details of upward of one hundred and forty interferometric examinations of patients at the Strasbourg Eye Clinic, mainly those suspected of having disturbances of internal secretions, ocular tuberculosis or neoplasms. His conclusions are as follows: Interferometry gives results which are, at times, disturbing in their exactness; at others, impressive in their indefinite character. It is a compli-

cated, delicate method which carries with it numerous sources of error. In spite of that and before deciding as to its value one way or another, there is every reason to continue the study of this method theoretically and practically.

The following papers were also read. No abstracts were furnished.

Anomalies of Chromatic Sensation. Dr. Polack, Paris.

Ptosis, Blepharochalasis and Fat Hernias of the Lids. Dr. Bourguet, Paris.

Akinesis. Dr. Kapuscinski, Posen.

Observations on the First Thousand Cases of Dacryocystorhinostomy (Lantern Projections). Dr. Gomez-Marquez, Barcelona.

Some Epidemics of Follicular Conjunctivitis in the Region of Algiers. Dr. Toulant, Algiers.

Comparative Study of Procedures for Intracapsular Extraction of the Cataractous Lens (Forceps, Erisiphak, Electrodiaphak) (Lantern Slides). Dr. Gomez-Marquez, Barcelona.

Our Results up to the Present in Treatment of Retinal Detachment by Diathermy (Lantern Slides). Dr. Jeandelize and Dr. Baudot, Nancy.

Transitory Ophthalmoscopic Picture of Retinitis in the Course of a Syndrome of Intracranial Hypertension. Dr. Pesme, Bordeaux.

Remarks on the Functional Examination of the Retinal Circulation. Dr. Dubar, Paris.

Case of Symmetrical Lymphadenoma of the Orbit (Lantern Slides). Dr. Kapuscinski, Posen.

Malignant Tumor (Lymphocytoma) of the Orbit in a Newly Born Child (Lantern Slides). Dr. E. Kalt.

Ocular Troubles of Aviators. Dr. Worms and Dr. Rateau, Paris.

Sympathogonic Sympathoma of Suprarenal Capsule. Secondary Manifestation in the Visual Apparatus (Lantern Slides). Dr. Van Der Straetten and Dr. Maldagne, Louvain.

Book Reviews

A Brief History of the Massachusetts Eye and Ear Infirmary and of the Howe Laboratory of Ophthalmology. Pp. 23, Boston: Privately printed, 1933.

The Massachusetts Eye and Ear Infirmary originated through the initiative of two young physicians of Boston, Dr. Edward Reynolds and Dr. John Jeffries, in 1824. Both had studied in London at Moorfields, founded some years earlier, in 1805, and were much impressed by what they saw there. In an address delivered in 1850, Dr. Reynolds stated that he had been influenced in the founding of the Massachusetts Eye and Ear Infirmary by the fact that one of its founders (himself) had the happiness of restoring a beloved father's sight by an operation for cataract. The tender relationship in this case of the surgeon and patient became extensively known among the small population then comprising the community; it brought to his observation a large number of patients with diseases of the eye and soon revealed the fact that the poor and laboring classes are peculiarly liable to these diseases—a fact now familiar to every one acquainted with the records of ophthalmic dispensaries.

The hospital was first a small building sufficiently large for ambulatory patients, bed patients being boarded out in nursing homes in the neighborhood; practically all the patients were of the former class. Larger quarters were obtained in 1836, and accommodations were provided for 20 beds. The dispensary cases at that time comprised about 2,000 annually. A further move was necessitated in 1894; this site was maintained until 1899, when the building now occupied was erected. A building for contagious diseases of the eye was added in 1901, and a pathologic laboratory in 1903. A complete optical department was provided for the first time in 1914. In 1871 a department for diseases of the ear was created, and in 1928 the laryngologic department of the Massachusetts General Hospital was taken over by the Infirmary. The Infirmary has become an institution of 231 beds, with an attendance in 1932 of 7,596 inpatients and 99,646 outpatients.

Brief biographic sketches of a number of the most distinguished members of the staff follow, including those of Drs. Edward Reynolds; John and Benjamin Joy Jeffries, father and son; George E. Bethune, John H. Dix, Hasket and George S. Derby, father and son; Oliver F. Wadsworth, Myles Standish and Alexander Quackenboss. With the exception of Dr. Dix, all had studied for some time abroad. All became members of the American Ophthalmological Society, Dr. Reynolds and Dr. Jeffries having been the founders of that society. These and their associates in the Infirmary were responsible for the high grade of excellence attained by ophthalmologists in New England and contributed much toward creating and maintaining the high standard of ophthalmology in America. With its two fellow pioneer ophthalmic institutions in New York and Philadelphia, the Massachusetts Eye and Ear Infirmary has not only given medical and surgical relief to countless thousands but has provided instruction for hundreds of neophytes in the science of ophthalmology.

This period of a century marked not only the establishment of the three great hospitals for the treatment of the eye in Boston, New York and Philadelphia but also that of the three famous schools for the blind in the same cities. All well maintained during the past hundred years, these schools have graduated hundreds of pupils who have been aided in overcoming their visual handicap and whose lives have been made useful and happy thereby. Truly that was a beneficent period, and recognition should be given to the fact. In the Massachusetts Eye and Ear Infirmary organized classes in the diagnosis and treatment of diseases of the eye and in the handling of patients with such diseases have been offered to physicians since 1836, to nurses from affiliated training schools since 1875 and to social workers specializing in this field since 1907.

The Howe Laboratory and Library were established in 1926 by the gift of Dr. Lucien Howe of Buffalo of $500,000 to the Harvard University Medical School, the income to be used for research in ophthalmology. Later, according to the terms of Dr. Howe's will, $240,000 was added, the Infirmary providing space to house the Howe Laboratory of Ophthalmology. The Howe Library was donated by Dr. Howe, who gave his valuable collection of books and periodicals to it; Mrs. Howe supplemented this gift by many valuable books of recent publication. Dr. Howe's gift provided for public lectures under the auspices of the Howe Laboratory, two of which have already been given. The brochure contains a brief biographic note of Dr. Howe in which he is cited as the author of the New York State Howe Law which made prophylaxis of the eyes of the new-born compulsory. It also refers to him as being the author of a standard textbook on the ocular muscles. After studying medicine at Harvard University Medical School and Bellevue Hospital Medical College, he pursued his studies abroad for a considerable period, returning to practice ophthalmology in Buffalo. In this he was largely successful. His was a scientific mind, and he was ever bent on research. The Howe Laboratory gave final expression to his ambition. He spoke many languages fluently and was conversant with the foreign ophthalmic literature. A familiar figure at all national and international congresses, he did much to forward ophthalmology in America and to cement friendships with foreign colleagues, begun in his student days.

WILLIAM CAMPBELL POSEY.

Seventh Annual Report of the Giza Memorial Ophthalmic Laboratory, Cairo. Pp. 140. Cairo: Schindler's Press, 1933.

The Giza Laboratory is still under the able direction of Rowland P. Wilson; Major F. H. Stewart is the pathologist, while Ahmed Fahmi Abbassi and Abdel Fattah El-Tobgy are assistant pathologist and assistant surgeon, respectively, on the permanent staff.

The usual postgraduate instruction was given during 1932, after receiving which candidates for service in the ophthalmic section of the Public Health Service had to pass a primary and a final examination. It is noted that physicians who are graduates of the Faculty of Medicine of the University of Cairo possess a far better knowledge of ophthalmology than those graduated abroad. This may be due to the fact that

their education is determined by ophthalmologic features peculiar to Egypt and not necessarily to the fact that a poorer training is received abroad.

The Giza Laboratory functions chiefly as a central pathologic laboratory for the 52 government ophthalmic hospitals in Egypt, which admit approximately half a million new patients annually. During 1932 there were 122 enucleations, as compared with 150 in 1931. Sixty-six per cent of the 122 enucleations were performed on eyes rendered blind as a result of purulent ophthalmia. (In 1927, 77 per cent of the enucleations were attributable to this cause.) It is believed that only about 1 per cent of all cases of blindness in Egypt are due to uncomplicated trachoma.

Four hundred and seventy-two pathologic specimens examined during 1932 are tabulated in the report. In addition, it indicates that over 2,000 bacteriologic, hematologic, helminthologic and other special examinations were carried out for the Government Hospital at Giza.

Sixteen uncommon tumors are briefly described, all the descriptions being accompanied by photomicrographs.

In the clinical section of the report a list of the more interesting cases seen during 1932 is included, together with short abstracts of a few unusual cases, which are illustrated either in black and white or in color.

Attention is called to the fact that retinitis pigmentosa comprises about 8 per cent of the diseases of the retina and choroid. Many patients also have night blindness without retinal changes, and it is suggested that the anemia produced by infestation with Ancylostoma may be a contributing factor.

High myopia is frequently seen, which is the more striking because most of the Egyptian patients are illiterate. Hereditary weakness of the posterior part of the sclera is suggested as a possible causal factor.

Clinical and therapeutic research has been along two lines: the control and prevention of trachoma (which has an incidence of nearly 100 per cent) and the investigation of the Prowazek-Halberstaedter inclusion bodies found in trachoma.

With regard to the prevention of trachoma, 5 new-born infants in Bahtim were treated regularly with acriflavine hydrochloride 1 : 5,000 in castor oil. Trachoma developed in all within from four to nine months. Five other new-born infants received 0.5 per cent zinc sulphate in 0.01 per cent mercury pyrotannate regularly. Trachoma developed in 1 after nine months, while the other 4 were still free from trachoma after a year of treatment.

Various methods of treatment have been used for combating existent trachoma. A solution containing 45 per cent chaulmoogra oil and 10 per cent copper sulphate applied to the lids weekly for eight weeks seemed of definite value, though this treatment is drastic.

Sterilized pure oil of Hydnocarpus wightiana with 4 per cent creosote, which was alternated every two days with 10 per cent copper sulphate, for eight weeks seemed to be of definite value in producing smoothly cicatrized lids.

Thermotherapy seemed to be of benefit in cases of pannus but was of little or no value for palpebral lesions.

Ointments containing antimony were of no value, nor did polyvalent mixed vaccines have any beneficial effect on trachoma in any of its stages.

Bitot's spots were made to disappear, after a preliminary scraping of the affected areas, by the application of pure phenol. Usually, one treatment was sufficient.

The report closes with an article by Dr. F. H. Stewart, who states in his summary that the Prowazek-Halberstaedter inclusion bodies found in the conjunctiva, especially in trachoma, are apparently due to the phagocytosis of pathogenic bacteria by epithelial cells, and that they do not occur in uncomplicated trachoma but are due to a secondary bacterial infection.

The complete report is an able summary of the useful and highly commendable work which is being done at the Giza Laboratory by Dr. Wilson and his associates.

W. F. DUGGAN.

Individual Differences in Normal Colour Vision. A Survey of Recent Experimental Work (1910-31). By W. O'D. Pierce. Price 2s. net. Pp. 96. London: His Majesty's Stationery Office, 1933.

Recent experimental work in vision has established the fact that among "normal" subjects many individual differences in the perception of color occur—that under the same experimental conditions, different subjects give significantly different results. This survey is limited to individual differences between color "normals" (some results from mixed and color blind groups are given in the last section), and it deals with experimental work only. Its aim is to give a review that will make this information available to research workers on color vision. The period from 1910 to 1930 has been covered, and the terminology recommended in the 1922 Report of the American Optical Society's Committee on Colorimetry is used.

One hundred and one articles are reviewed, some at length, and there is a complete bibliography. The object of the author's survey has been attained admirably, and his work contains in a small compass an amazing amount of information on a fascinating subject.

JOHN B. LYNCH.

Diseases of the Eye. By M. S. Mayou. Fourth edition. Revised and Rewritten by F. Ripley and A. Sorsby. Cloth. Price, $2.25. Pp. 260, with 38 illustrations. London: Oxford University Press.

This is a small, handy volume of 249 pages which is intended to serve as an introduction to ophthalmology for the student. Its popularity is shown by the fact that it is already appearing in a fourth edition, and the present editors, aware of the close association of ophthalmology with general practice and with the basic sciences, have brought the subject matter well up to date.

This little volume is quite complete, but it suffers by too great brevity, particularly in the fundamentals, which are so important to the beginner; one misses also the great help of illustrations, of which there are only 38. The book, therefore, will be of greater service to the general practitioner or the advanced student.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217, Faubourg St. Honoré, Paris.
Secretary-General pro tem: Dr. A. Churchill, 66 Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Frank H. Crawley, 5 Fitzwilliam Pl., Dublin.

Ophthalmological Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay, 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. A. C. Hudson, 50, Queen Anne St., W. 1, London.
Secretary: Dr. J. D. Magor Cardell, 27, Weymouth St., W. 1, London.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Time: 1934.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. William C. Finnoff, 1612 Tremont St., Denver.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: Cleveland. Time: June 11-15, 1934.

American Academy of Ophthalmology and Otolaryngology

President: Dr. J. M. Wheeler, 30 W. 59th St., New York.
President-Elect: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Sept. 9-14, 1934.

American Ophthalmological Society

President: Dr. W. G. M. Byers, 1458 Mountain St., Montreal.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Lucerne, Que. Time: July 9-11, 1934.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan.
Managing Director: Mr. Lewis H. Carris, 450, 7th Ave., New York.
Place: New York. Time: November, 1934.

SECTIONAL

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Hugo B. C. Riemer, 128 Newbury St., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. A. W. Morse, 66 W. Park St., Butte, Mont.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Butte, Mont. Time: July 16-18, 1934.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Francis A. Brugman, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Noble Dick, 1115 Terry Ave., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Harry Shearer, 405 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. W. S. Beyer, 1101 Talcott Bldg., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President, Dr. A. R. McKinney, 703 Second National Bank Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Dunbar Roy, 113 N. Pryor St., Atlanta, Ga.
Secretary: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Place: New Orleans. Time: 1934.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Carl Snapp, 604 Medical Arts Bldg., Grand Rapids.
Secretary-Treasurer: Dr. Lee O. Grant, 420 Medical Arts Bldg., Grand Rapids.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. O. G. A. Barker, 532 Main St., Johnstown.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.
Time: Third Thursday of October and May.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 920 Republic Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Edward J. Whalen, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. W. R. Bedingfield, 753 Broad St., Augusta.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.
Place: Augusta. Time: May, 1934.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Fred Overman, 611 Hume-Mansure Bldg., Indianapolis.
Place: Richmond. Time: Dec. 12, 1934.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.
Secretary-Treasurer: Dr. Lawrence A. Taylor, 118 E. Second St., Ottumwa.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Peter T. Grant, 148 Monroe Ave., Grand Rapids.
Secretary: Dr. Ralph B. Fast, 1410 Bank of Kalamazoo Bldg., Kalamazoo.
Place: Battle Creek. Time: 1934.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Gordon B. New, Mayo Clinic, Rochester.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. S. T. Hubbard, 294 State St., Hackensack.
Secretary: Dr. G. S. Laird, 125 Central Ave., Westfield.
Place: Atlantic City. Time: June, 1934.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.
Secretary: Dr. Webb W. Weeks, 20 E. 53rd St., New York.
Place: Utica. Time: May 14-16, 1934.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. O. Ruud, 318 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.
Place: Valley City. Time: 1934.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Robert C. O'Neil, 105 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Walter Bristow, Medical Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. D. Blassingame, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. Ralph O. Rychener, 130 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. C. C. Cody, 1304 Walker Ave., Houston.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY
President: Dr. F. R. Slopanskey, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welsh, 920 Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY
President: Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.
Secretary: Dr. Edwin W. Burton, 104 E. Market St., Charlottesville.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION
President: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.
Secretary: Dr. W. F. Beckner, 1050 Fifth Ave., Huntington.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Browne Morgan, 260 Liberty St., Bloomfield.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ACADEMY OF MEDICINE OF TORONTO, SECTION OF OPHTHALMOLOGY
Chairman: Dr. Colin Campbell, 170 St. George St., Toronto.
Secretary: Dr. H. M. Macrae, 170 St. George St., Toronto.
Time: First Monday of winter months.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. W. L. McDougall, 50 Whitehall St., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY
Chairman: Dr. Benjamin Rones, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY
President: Dr. W. F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Secretary: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB
President: Dr. H. H. Glosser, 448 Franklin St., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.

Chicago Ophthalmological Society

President: Dr. Dwight C. Orcutt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. A. L. Stotter, 1148 Euclid Ave., Cleveland.
Secretary: Dr. James T. Collins, 1105 Medical Arts Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. W. P. Chamberlain, 7405 Detroit Ave., Cleveland.
Secretary: Dr. A. D. Ruedemann, Cleveland Clinic, Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. M. Goldberg, 328 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. T. S. Love, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Daniel Brannin, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. C. Walker, 410 6th Ave., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President, Dr. Edward B. O'Keefe, 901 State St., Schenectady.
Secretary-Treasurer: Dr. W. C. Mott, 118 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Valin R. Woodward, Flatiron Bldg., Forth Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 320 Medical Arts Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Secretary: Dr. B. F. Payne, 1304 Walker Ave., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. D. A. Bartley, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. M. B. Simpson, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Clifford S. Losey, 219 E. 10th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Kenneth C. Brandenburg, 707 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. F. H. Brandt, 1136 W. Sixth St., Los Angeles.
Secretary-Treasurer: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Charles Dwight Townes, 332 W. Broadway, Louisville, Ky.
Secretary-Treasurer: Dr. J. S. Bumgardner, 1103 Heyburn Bldg., Louisville, Ky.
Place: French Village. Time: Second Thursday of each month, September to May, inclusive, at 6:30 p. m.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. George B. Trible, 1801 I St., Washington.
Secretary: Dr. Harry F. Davies, 1835 I St., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month.

Montreal Ophthalmological Society

President: Dr. J. P. E. Bousquet, 66 Sherbrooke St., West, Montreal, Canada.
Secretary: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Frederick E. Hasty, 1), 7th Ave. N., Nashville, Tenn.
Secretary-Treasurer: Dr. Kate Savage Zerfoss, 803 Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Webb William Weeks, 20 E. 53rd St., New York.
Secretary: Dr. Guernsey Frey, 121 E. 60th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. Abbott M. Dean, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. Robert H. Rasgorshek, 425 Aquila Court, Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Secretary: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Time: Second Tuesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Lee, 501 E. Franklin St., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. W. D. Edwards, 389 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. E. C. Spitze, 234 Collinsville Ave., East St. Louis, Ill.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Robert E. Parrish, 712 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. King Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert S. Irvine, 490 Post St., San Francsico.
Secretary: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December

Shreveport Eye, Ear, Nose and Throat Society

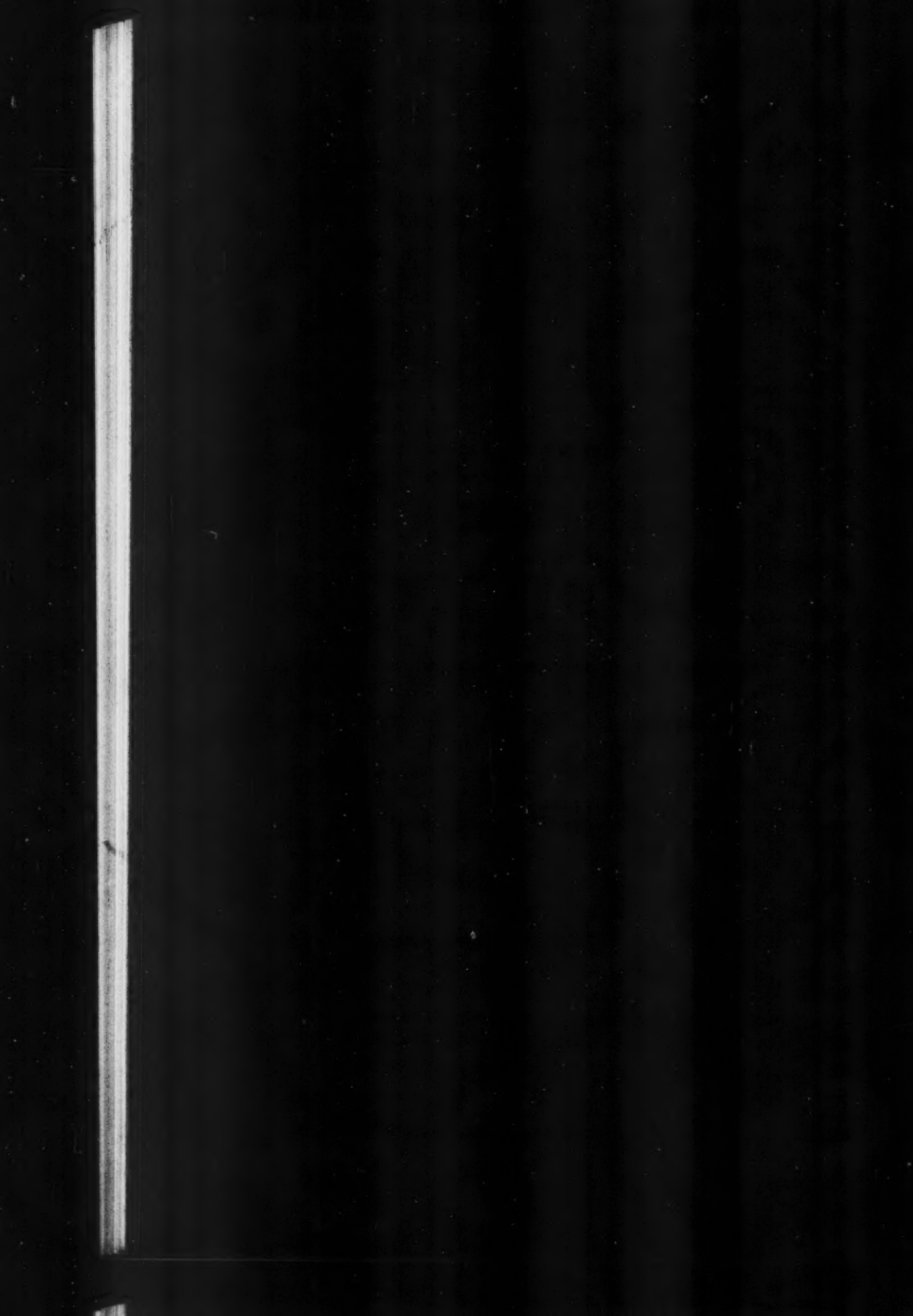
President: Dr. T. D. Boaz, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: 1240 Texas Ave. Time: 8:15 p. m., fourth Tuesday of every month except June, July and December.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. A. Veasey, Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. J. L. Walter, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. H. O. Brust, 820 S. Crouse Ave., Syracuse, N. Y.
Secretary-Treasurer: Dr. W. J. Werfelmann, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, Medical Arts Bldg., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, Medical Arts Bldg., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.